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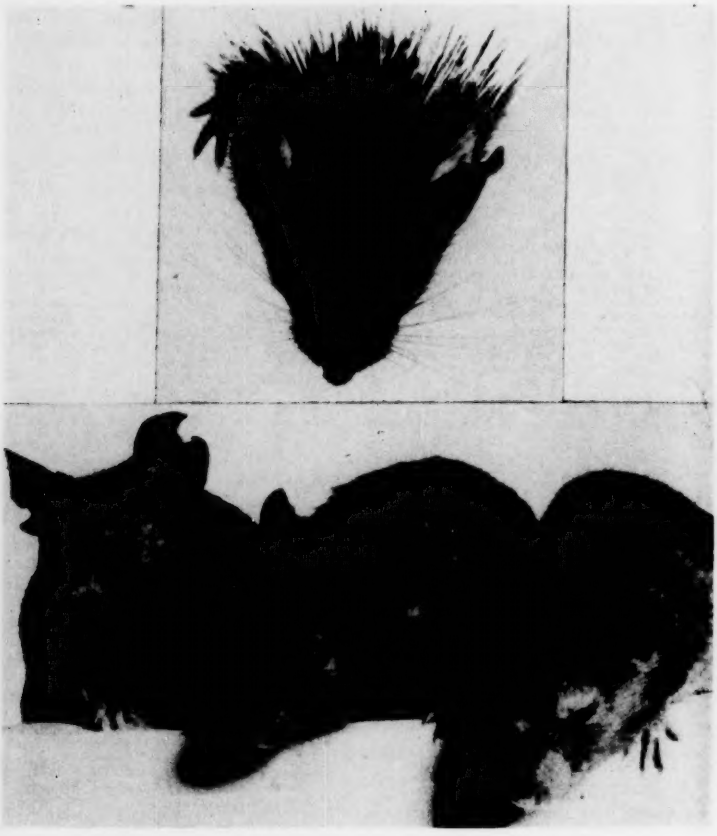

WHISKER-EATER AND CLOSE-SHAVED "VICTIMS"

(SEE PAGE 77)

The Coy-Dog: A Problem-Hybrid

"Double-Muscled" Cattle

Factors Affecting the Human Sex Ratio

VOLUME 43 MARCH-APRIL, 1952 NUMBER 2

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An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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MUSCLE HYPERTROPHY COMPARED WITH NORMAL

Figure 1

A—shows bull with muscular hypertrophy compared with a normal bull of the same age raised under similar conditions. Note the peculiar stance of the hyper-muscled animal, which is typical. Also note the groove running down the side of his hind leg. These are characteristics of the individuals showing this peculiarity.

MUSCULAR HYPERTROPHY IN CATTLE*

J. F. Kidwell, E. H. Vernon, R. M. Crown and C. B. Singletary†

AN anomalous condition, muscular hypertrophy, appeared in 1950 and 1951 in the crossbred Africander-Aberdeen Angus line at the Iberia Livestock Experiment Farm at Jeanerette, Louisiana. This herd originated in 1934 through crossing a purebred Africander bull, 1H (Figure 2), with purebred Aberdeen Angus females. In 1937 male 1H was replaced with male 2H.

As the major objective was to develop a distinct line adapted to Gulf Coast conditions, intense matings of F_1 and succeeding generations have been practiced. The line has been maintained as a one, two or three sire herd, depending upon the circumstances. Inbreeding has been kept to a minimum insofar as was practicable within the breeding plan. The average coefficient of inbreeding of all animals now in the line is only 0.68 percent.

In 1949 part of the herd was transferred to the Louisiana Agricultural Experiment Station at Baton Rouge and part to the North Louisiana Agricultural Experiment Station at Calhoun. The gene determining red coat color was deliberately introduced in the original matings, and the red segregates were transferred to Calhoun. Animals sent to Baton Rouge were ⅜ Africander and ⅝ Angus, and those remaining at Jeanerette were ½ Africander and ½ Angus as calculated by "percentage of blood." These animals were solid black. Five anomalous calves (males 236H7, 246H3, 11, and females 8, 3H7) were born at Baton Rouge and at Jeanerette (Figure 2). These individuals were used in describing the anomaly and analyzing its mode of inheritance.

Review of Literature

Weber and Ibsen[6] described as "double-muscled" an anomaly which appeared in a herd of purebred Hereford cattle. The individuals were abnormally thick and full in the thighs, with deep grooves between the muscles. In some the rump drooped with the twist lacking depth and fullness. The carcass of one animal was examined and found to correspond closely to a type known to meat packers as "Yorkshire." This type is also known by other descriptive colloquialisms, such as "Teeswater." Carcasses are characterized by a scant covering of fat over the round and a general lack of marbling. The meat has a tendency to be coarse grained.

Weber and Ibsen presented evidence that the double-muscled character exists in all purebred beef breeds of European origin in the United States, although it varies considerably in expression. On the basis of their data, they suggested that the character might be conditioned by a recessive gene, but concluded that the mode of inheritance was not satisfactorily established, particularly with reference to modifiers.

Wriedt[7] stated that the same anomaly has been known in Germany as "Doppellender" since 1888 and has occurred widely in different breeds in various European countries. Wriedt emphasized the variable expression of the character. He concluded that it was due to an incompletely dominant gene and that homozygous dominants, male and female, are either sterile or at least poor breeders. Heterozygous individuals were said to be completely fertile. Although he did not describe these in detail, he reported that some appeared to be entirely normal.

Kronacher[2] was inclined to consider the mode of inheritance as quantitative, but concluded that it was not completely explained. Black[1] states, "This character causes a doubling of the muscles in the hindquarters and loin and is inherited as a recessive." According to Schaper[4] "This is superfluous development of the loin and shank. . . . Such calves are in general infertile. Investigations show

*This study was conducted in cooperation with the Bureau of Animal Industry, U.S.D.A., under the Southern Regional Project S-10 on Beef Cattle Breeding Research. The authors are indebted to Mr. Aime Fortier of the Iberia Livestock Experiment Farm, for assistance in the conduct of this investigation.

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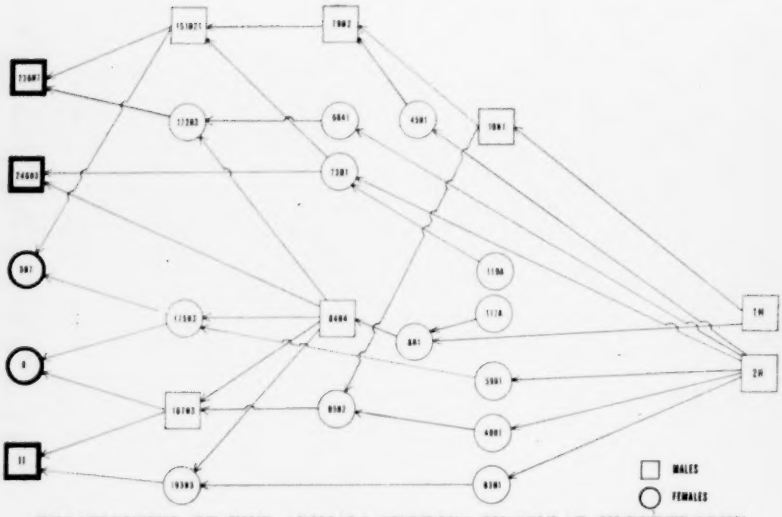


RELATIONSHIP OF FIVE ANIMALS SHOWING MUSCULAR HYPERTROPHY

Figure 2

This network pedigree shows the relationship of the three affected males, 236H7, 246H3, and 11, and females 3H7 and 8.

that this deformity is doubtless inherited but its mode of inheritance is not clear."

Description of Anomaly

Externally, the anomalous calves in the Africander-Angus herd at Jeanerette and Baton Rouge closely resemble the animals described by Weber and Ibsen. Thighs are extremely thick and full, with a deep groove between the vastus lateralis and semimembranosus muscles. The groove appears to be due to hypertrophy of these muscles, which lie immediately under the biceps femoris. The twist lacks depth and fullness because muscling does not extend toward the hocks. Excessive muscling over the withers exists in older animals. Anomalous individuals assume an unusual stance, with fore and rear legs extended anteriorly and posteriorly. In this position they are distinctly sway-backed and weak in the loin. However, muscular coordination appears normal.

In two instances, dams experienced difficulty in parturition; one died later as a result of calving injuries. Schaper stated that difficulties of parturition are common with this anomaly, dismemberment of the calf in utero frequently being necessary.

In order to study anatomical deviations of the anomaly a normal and an abnormal bull calf was slaughtered April 3, 1951. The anomalous calf, male 246H3 (Figure 1), born at Jeanerette, February 24, 1950, and transferred to Baton Rouge, December 18, 1950, was maintained on pasture with a normal calf of comparable age until slaughtered. The animals appeared to be in similar physical condition, each weighing approximately 500 pounds.

Photographs of these animals, half of each carcass and a cross section through one round of each, are shown in Figures 3 and 4.

One half of each carcass was dissected. No actual doubling or duplication of the muscles was observed. In the anomalous male there was an obvious abnormal enlargement of the thigh and loin muscles associated with a displacement of the muscle belly toward the origin. This was also true to a lesser degree of the muscles in the area of the withers and brisket. The condition appears to be a case of muscular hypertrophy rather than muscular duplication. In this case the term "double-muscled" is a misnomer. The authors prefer the term "muscular hypertrophy."

Macroscopically the muscle appeared to be of a coarser texture than normal with more distinct longitudinal connective tissue septa. However, microscopic examination did not reveal gross histological abnormalities. In the anomalous animal, there was practically no subcutaneous fat over the round and loin and very little caul fat or kidney fat. The slight amount of fatty tissue present in the region

ANATOMY OF MUSCLE HYPERTROPHY
Figure 3

Left half of carcasses of hyper-muscled (*A*) and of normal bull (*B*) shown in Figure 1. These animals are sometimes erroneously called "double muscled." There is no duplication of muscles; only a considerable overgrowth. Animals displaying this peculiarity are probably homozygous for a gene showing some effect in the heterozygote. The homozygous form is undesirable from the commercial point of view and appears to be of low fertility. The heterozygote may be selected as representing a superior type.

of the kidneys was abnormally soft and degenerate.

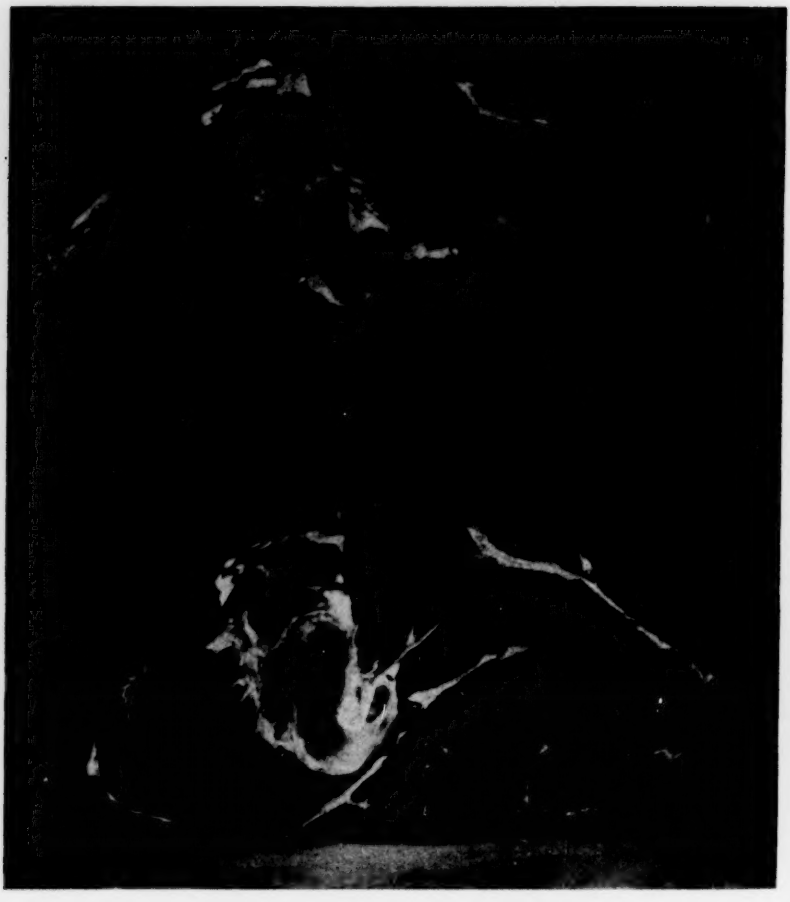
Weber and Ibsen reported that the general lack of external fat resulted in excessive drying and consequent undesirability of the carcasses of anomalous animals. Shrode and Lush[5] cited some verbal reports from the trade that the meat is tender, richly flavored and highly esteemed by connoisseurs. One local packer indicated a preference for anomalous animals for veal, but all others queried considered them inferior.

The carcass of the anomalous animal studied by the authors dried excessively. Cooking and palatability tests on comparable cuts from the two calves did not reveal differences in tenderness or palatability.

Examination of the brain revealed unusual pigmentation and less vascularization than would be expected in the pia mater in the frontal lobes extending posteriorly along the dorsal median sulcus. This condition was not necessarily bilateral, but was confined to one or the other of the hemispheres. No appreciable difference was noted in brain weight between the normal and anomalous calves.

Complete genital tracts of both animals were removed and examined.* In the anomalous animal a complete adhesion of the glans penis to the sheath extending posteriorly for a distance of about two inches completely enveloping the penis was found. The adhesion of the penis and sheath could be extended from the body approximately two inches. This male was

*The authors are indebted to Dr. Cecil Branton, Louisiana State University Dairy Department, who made the examination.



HYPERTROPHIED MUSCLES IN SECTION

Figure 4

Section through the round of hyper-muscled (above), and normal bull (below). It is generally maintained that the flesh of the "big-muscled" animals is coarser than normal; it definitely lacks fat. Though the authors could not detect differences in tenderness or palatability, excessive dryness was noted.

incapable of copulation and for all practical purposes was sterile. Semen might have been procured artificially only by means of a special massage technique.

The adhesion may have been due to an infection or to hypoplasia of the glans penis and hyperplasia of the sheath. As infections of the glans penis are infrequent in young males, and there was no indication of infection in this animal, the authors regard the adhesions as the result of underdevelopment of the glans penis and hyperplasia of the sheath.

The diameter of the penis was approximately two-thirds that of the normal male, and the testicular weight of the anomalous animal was approximately 60 percent that of the nor-

mal individual. In the absence of a larger control series it was impossible to determine whether these sexual organs were below normal range for males of this age and weight. More spermatozoa were evident in the tract of the normal male, and exhibited greater motility. Difficulty was encountered in obtaining spermatozoa from the tract of the anomalous male due to the limited quantity of semen. A large proportion of dead and abnormal spermatozoa was found in the semen samples. This further suggests a low fertility in the anomalous male.

Another anomalous male, 236H7, was sold for use as a herd sire. This animal was last examined by the authors April 10, 1951. The owner reported that the bull had bred a 15-month-old heifer. This animal had been on full feed for some time and was well developed. Testicles and glans penis were of normal size. It was determined by massage that the individual was capable of ejaculation and no gross abnormalities of the copulatory organs were found.

From these limited observations it appears that a reduced fertility or even sterility of the males may be associated with muscular hypertrophy. It appears possible that anomalous males may be entirely fertile. No data exist on the fertility of the anomalous female at Baton Rouge. An attempt will be made to breed her to male 236H7.

Mode of Inheritance

Muscular hypertrophy appeared *de novo* in the experimental Africander-Aberdeen Angus herd during 1950 and 1951. A previous appearance would almost certainly have been noted. Occurrence of a new mutation is not likely as the five anomalous animals were by three sires out of four dams. Therefore, it seems reasonable to conclude that the character is not conditioned by a dominant gene. Every animal in the herd has been examined. A few animals known to be carriers appear entirely normal, but most of them exhibit evidence of muscular hypertrophy in varying degrees. This is evidenced by a relatively wider and more muscular-appearing loin and a wider, fuller round than in normal animals. The characteristic groove between the thigh muscles is usually prominant. Animals which may or may not be carriers could be divided into two distinct groups when judged on this basis. This strongly suggests that the gene acts as an incomplete recessive with variable expressivity. According to this hypothesis the heterozygote would range in appearance from completely normal to a form resembling the homozygous recessive. There is some variation among homozygotes in the intensity with which muscular hypertrophy is expressed. Whether the variable expressivity is due to the action of modifiers or environment or to genetic-environmental interaction is not known. A pedigree network of the anomalous calves is shown in Figure 2. According to the author's hypothesis the anomalous animals are homozygous and the sire and dam of each is heterozygous.

As this herd is the result of the species cross, *Bos taurus* × *Bos indicus*, the question of the origin of the gene in the herd is of interest. Inspection of the pedigree limits the possibility to four foundation animals: purebred Angus females 117A and 119A and purebred Africander males 1H and 2H. The critical pedigree is that of male 246H3, tracing through heterozygous male 84H4 to female 117A and male 1H; on the dam's side through heterozygous female 73H1 to female 119A and male 2H. Therefore, either female 117A or male 1H and either female 119A or male 2H were heterozygous. It is not likely that an Africander and an Angus were both heterozygous. Had that been so, the anomaly should have appeared earlier. It seems probable that both purebred Africander males or both purebred Angus females were heterozygous.

The pedigree for male 1H is complete only for the sire and dam. This animal was born November 27, 1932, and was loaned to the Iberia Station from March 21, 1934 to October 7, 1937. Male 2H was born June 5, 1930, and was imported from South Africa in 1932 by the King Ranch. He was received at the Iberia Station April 28, 1937. The pedigree is complete through four generations. Information available does not indicate any relationship between these bulls.

The two purebred Angus females were purchased from a Kansas breeder. It was not possible to obtain pedigrees for these animals. However, they were born in the same herd in the same year, and it is likely they were related. Since the gene determining muscular hypertrophy has been reported in the Aberdeen Angus breed, it seems likely that these two females were heterozygous and that they introduced the gene into the herd.

Discussion

The hypothesis of an incomplete recessive with variable expressivity appears to agree with our observations and data as well as those of Weber and Ibsen. Weber and Ibsen questioned Wriedt's hypothesis of an incomplete dominant because the latter stated that homozygous dominants are either sterile or are poor breeders. However, Weber and Ibsen observed two anomalous males which sired 167 calves out of normal cows over a five-year period. Nineteen calves from 10 dams were anomalous. They reasoned that under Wriedt's hypothesis the two males must have been heterozygous and that approximately

one-half the calves should have been anomalous. If some of the homozygotes are fertile these conclusions are unnecessary. Wriedt pointed out that homozygotes may be fertile, and the authors' observations as well as those of Weber and Ibsen are in agreement. The authors prefer to classify the gene as an incomplete recessive with variable expressivity, rather than as an incomplete dominant.

Lush[3] suggested that the heterozygotes may have a selective advantage over homozygous normals. Shrode and Lush concluded: "Double muscling appears to be an example of a characteristic kept in the breeds but at a low frequency, because the heterozygote is preferred over both homozygotes but one homozygote is very much preferred over the other. The almost universal desire of beef breeders to increase the thickness of muscling in their cattle leads them to select for breeding many of the heterozygotes which manifest this double-muscling to only a slight or moderate degree. But the abnormal appearance and partial sterility of those which manifest the character extremely lead to intense selection against them. At least this conjecture is plausible, although other explanations can hardly be excluded as long as the genetics of double-muscling remains uncertain."

These data appear to support such a conclusion. In the second generation a heterozygous male, 84H4, was selected as a herd sire and only heterozygous sires have been used to any appreciable extent thereafter. The frequency of the gene is estimated roughly as 0.28 by use of the Hardy-Weinberg binomial and the proportion of anomalous calves born in 1950 and 1951. If it is assumed that the gene was introduced by Angus females 117A and 119A, the original frequency in the herd can be estimated at 0.04. There has been an increase of frequency of 0.24 in approximately three generations, or an average increase of 0.08 per generation.

The present high incidence of the gene could be due to genetic drift, or chance. It might also be explained on the basis of selective correlation, i.e., the original heterozygous animals contributed other desirable genes which caused a high proportion of their progeny to be retained in the breeding herd. In this study it was not possible to evaluate the influence of these factors. The slight modification of body proportions observed in the heterozygotes is considered highly desirable by most animal husbandmen. This provides a basis strongly suggesting the possibility of a selective advantage of heterozygotes.

Homozygotes are undesirable commercial animals due to lack of external fat and high frequency of breeding and parturition difficulties. This appears to be a case in which animals that are heterozygous for a deleterious gene may have a selective advantage over homozygous normals.

Summary

A muscular anomaly of cattle, muscular hypertrophy, appears to be inherited as an incomplete recessive with variable expressivity. Although the homozygote is an undesirable commercial animal, the heterozygote appears to be favored by selection.

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STUDIES ON THE HUMAN SEX RATIO

Relation of Time of Conception During Estrous Cycle to the Sex Ratio

MARIANNE E. BERNSTEIN*

LONG before the X-Y chromosome mechanism of sex determination was discovered, biologists speculated that the moment during the estrous cycle at which fertilization takes place has a bearing on the sex ratio. The French biologist Thury in 1864[12] wrote that a friend of his by the name of Cornaz, observed that when his animals were serviced early in heat he got 22 female offspring in succession as against nine males in succession when serviced late in heat. In this country, F. L. Russell,[8] then director of the Maine Agricultural Experiment Station, reported obtaining 32 male calves and 51 female calves when he serviced his cows early in heat as against 42 male and 34 female calves when he serviced late in heat. While also at the Maine Agricultural Experiment Station, the famous biologist Raymond Pearl became interested in the results obtained by Russell and decided to extend these studies. In his first paper (1913) Pearl substantiated the findings of Russell that servicing early in heat resulted in a lower sex ratio than servicing late in the heat period; however Pearl points out that the data were not yet large enough to make the results statistically reliable beyond doubt. He therefore continued these experiments for a few more years, publishing his final results in 1917.[7]

This time his results were statistically reliable beyond any doubt. Pearl writes: "Some earlier statistics seemed to indicate that there is a possibility of influencing the sex ratio by controlling the time of service at different parts of the heat period. However, with more extensive statistics at hand, it appears conclusively established that there is no definite permanent relation between the time in the heat period at which the cow is serviced and the sex of the offspring. The apparent relationship between these two factors, which is believed by many breeders to exist and which our earlier statistics appeared to indicate, seem now to be purely accidental, and to have arisen only because of the comparative meagerness of the statistics on which the matter was discussed."

Pearl's final data were as follows: The sex ratio of 392 calves whose mothers were serviced early in heat: 51.0 percent male; the sex ratio for 522 calves whose mothers were serviced in the middle part of the heat period: 51.7 percent male; the sex ratio for 399 calves whose mothers were serviced late during the heat period: 46.9 percent male. A standard error for samples of size $N = 400$ is 2.5 percent, so that all deviation observed by Pearl for his animals is purely due to chance.

Other Data

That the time during the estrous cycle at which conception takes place has a bearing also on the sex ratio of man has been claimed by the German scientist Siegel. But here again the set of data were too small to give reliable results. Soon after Siegel's paper appeared, similar studies on much larger material were made by Fuerst, Prylls and others, and these scientists disproved Siegel's theory conclusively (as quoted by Wedervang[13]).

Recently, the theory of Thury has been retested in this country by Hart and Moody[5] on rats. These authors cite certain statistics on the sex ratio in man which, they believe, bear out their contention that the time during the menstrual cycle at which conception takes place has an effect on the sex ratio of the children.

Drs. Seymour and Koerner[10] reported that in a group of 9,489 births resulting from artificial insemination, a sex ratio of almost 150 boys to 100 girls was obtained. Hoelzel and Carlson[6] have tried to explain this high sex ratio by pointing out that the semen was mainly obtained from superior males who are known (Bernstein[1]) to have a high sex ratio. However, Folsome[2] together with a physicist, L. B. Tuckerman, pointed out certain errors in Seymour and Koerner's data, which make their reliability very questionable. Folsome, in a historical review of papers dealing with the

*Helen Putnam Fellow, Radcliffe College, Harvard University.

problem of artificial insemination, points out that in Seymour and Koerner's data this method gave approximately 23 times more success than that reported in the entire medical literature during the previous four decades. Tuckerman adds: "It may be too much to expect physicians to give a set of figures which will counterbalance, nonetheless it does seem that any comment on the unusual boy-girl ratios noted might better be withheld until the discrepancies in the figures are explained. Incidentally, there were no cases of twins reported in this article. . . . In an ordinary series of 10,000 pregnancies one would expect about 125 pairs of twins. Absence of any comment on this feature makes the reliability of the report seem even more doubtful. . . ."

Twin Sex Ratios

Next Hart and Moody cite certain statistics on multiple births in man which, they believe, show an excess of same-sexed fraternal twins and triplets. This "excess" is calculated on the assumption that 25.5 percent of human twin births are monozygotic, this percentage being derived from data by Guttmacher[4] and Greulich.[3] The differentiation by Guttmacher was made on the basis of placental counts; i.e. one placenta meant the twins were monozygotic, two placentas meant the twins were dizygotic. However, a study by Steiner[11] shows how erroneous this assumption is. Classifying 236 like sexed twins by the similarity method of genetic traits as mono- or dizygotic, Steiner found that 40 monozygotic twins had one placenta but that in addition 80 dizygotic twin pairs also had a single placenta. Of 116 twin pairs with two placenta, 16 pairs proved to be monozygotic and 100 pairs dizygotic.

The other study quoted by Hart and Moody on the frequency of mono- versus dizygotic twinning made by Greulich[3] did use the more reliable genetic similarity method. However, Greulich's series was not suitable for determining the proportion of monozygotic twins in the general population. Weinberg[14] pointed out many years ago that the death rate during the early years of life is higher among monozygotic twins. Greulich emphasized that his data were for that reason not suited to determine twin birth frequencies. Of the 495 propositi twin pairs, 204 were of grammar and high school age. Another 200 twin pairs were born in 1916 and 1917 so that they were in their late 'teens when he observed them. The remaining 91 twin pairs were infants, so that Greulich was unable to classify them by the genetic similarity method used with the older twins. The conclusion to be drawn from these rather confusing and conflicting points of view is that at birth identical twins must constitute considerably more than 25 percent of all twin-pairs born.

Nor can it be said that in humans there is an excess of like sexed fraternal triplet births. Hart and Moody cite data on the frequency of triplet births in the United States for the years 1941, 1942 and 1945. In all, there were 206 sets of three females, 202 sets of two females and one male, 188 sets of two males and one female, and 231 sets of three males. Assuming the incidence of male and female births in triplets to be about the same (there were actually 1,210 males and 1,271 females in the series, but this is a minor error which does not affect the conclusions) Mart and Moody computed a theoretical distribution of sex combinations in fraternal triplet sets as follows:

3 boys or 3 girls: $N(\frac{1}{2})^3$

2 boys and 1 girl, or 1 girl and 2 boys: $3N(\frac{1}{2})^2(\frac{1}{2})$

Although as many as 231 sets of three boys and 206 sets of three girls were observed, this computation of theoretical expectation of fraternal same-sexed triplets yields an expectation of only 103.5 sets. "There is thus a great increase in the number of like-sexed triplet sets . . . over the expected number."

However, Hart and Moody have assumed that all sets of triplets of unlike sex arise from three zygotes; there are, however, well known cases of unlike sexed triplets arising from two zygotes (Scheinfeld[9]). In that case, the expected number of sets of three males or three females (or either of the other two combinations) would be in the above mentioned case, 207 each. This is very close to the observed number of 231 sets of three males and 206 sets of three females. Therefore we only have to assume a logical proportion between the three types of tripling (i.e. from one or two or three zygotes) to obtain the observed number of triplet sets of equal sex, without claiming that sex determination is not at random in multiple human births.

Also from studies on certain dizygotic twin births it appears doubtful whether conception of dizygotic twins always takes place at the same time. In several cases involving disputed paternity of twins, it has been definitely established that the two twins had different fathers (Scheinfeld[9]).

Conclusions

1. On the basis of previous studies on the subject, it appears most unlikely that in higher mammals there is any relation between the time in the heat period at which conception takes place and the sex ratio of the offspring.

2. A critical statistical and genetic evaluation of human twin and triplet data presented by Hart and Moody, makes it appear highly unlikely that there is an excess of like-sexed fraternal twins and triplets in man.

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THE COY-DOG: HYBRID WITH A FUTURE?

UNDETERRED by the rigid ideas of geneticists, the press goes far afield in its experiments in hybridization. Perhaps this is because the city desk is not far from the composing room — the only place, those profound philosophers James Thurber and E. B. White once warned us, where the physical and the psychical can ever be safely mixed. The "succotash hybrid" — a smutted ear of corn claimed to be a cross between *Zea mays* and *Phaseolus limensis* — is a notable example of the Lamarkian power of this environment.

Nor are the fauna safe from the inventive inroads of the press. A favorite is the "moo-cow": the result of a definitely *excestious* alliance between a bull moose and a cow. The moo-cow's junior cousin, the perennial deer-cow hybrid also belongs in this category of hybrids unrecorded in the archives of science. Whatever the purchaser of a sirloin steak may suspect, the costliness of this commestible is not because it contains any dear (pardon the Freudian lapse, *deer,* of course) chromosomes. The parade of the circus side-show hybrids should also include the "turkhen," the cat-rabbit, and Hollywood's bastard progeny of apes and humans.

In the tradition of man-bites-dog classification of what makes news, a minor "flash" should be spread in the records of science: a hybrid recently in the news which actually seems to exist. Furthermore, this hybrid is highly newsworthy in another respect: it shows promise of becoming a population with a place in the ecology of our nation; perhaps useful, possibly sinister.

The hybrid in question is the "coy-dog."

That dogs do on occasion mate with their feral cousins has been a matter of rather lengthy record. Occasional references to coyote-dog hybrids appear in the literature. In his definitive monograph on the wolves of America, Dr. Goldman of the U. S. Biological Survey devotes considerable space to instances of varied detail and authenticity regarding the crossing of dogs and wolves.

Report on the Coy-Dog

Such hybrids were considered to be isolated oddities—mule-like in their lack of pride in—or hope for—posterity. But this assumption appears to have been based on inadequate evidence. In the Adirondack mountains in northern New York coy-dogs are even becoming numerous enough to form packs. They are being viewed with qualified alarm as a possible menace to agriculture.

Dr. Clayton Seagears, Director of Conservation Education of the New York State Department of Conservation, met the coy-dog on his home ground. In reply to an inquiry, Dr. Seagears writes as follows:

"There is strong evidence that coyotes (skulls and other characteristics exactly conforming to type) have bred with more or less wild dogs in this State for some time. Many individuals are being trapped by State trappers and others, shot by deer hunters and otherwise obtained which apparently show hy-

MATURE COY-DOG AND COY-DOG PUPS

Figure 5

The upper photograph shows a typical Adirondack coy-dog in summer fur. The ears are less slender than those of a true coyote. The nose pad is larger than in the coyote and the muzzle is dog-like. In the litter, one of the pups in the hole has a collie-like head and the pup just above it has white legs. Black and white spotted pups and self black pups have also been observed. The mother of these pups, as far as external appearance reveals, was a pure coyote.

bridization. We've had several litters dug from Adirondack dens in May and June which showed great variation in conformation and color.

"I got one litter of eight pups last Spring. Two appeared typical coyotes, four were spotted black and white, one had ears folded like a collie, the others were jet black all over. I now have alive a parti-colored bitch caught in a trap as a pup in the Adirondacks. Although we've had her three years, she remains just as wild as the day she arrived, and, except for coloring, her characteristics are coyotish except that her nose pad is too large and body structure is a bit too gross for a typical animal. We expect to mate her, probably in February, with a pure quill coyote from Missouri to see what happens. They've been together since last Spring. If anything happens, we'll be very glad to let you know. Incidentally, the mother of the pups I mentioned was trapped at the mouth of the den. She seemed to be pure coyote, as far as external characteristics permit any such identification.

I'm sending you two pictures which may be of interest. One is of a typical litter of what we call coy-dogs. These four were dug from a den in Lewis County, the Adirondacks, and show wide variation of form and color. Note the collie-like head of one of the pups in the hole as well as the white legs of the one just above it. The other picture is a typical Adirondack coy-dog in summer fur. Note that the ears are less slender than the true coyote's, that the nose patch is larger and that the muzzle is dog-like."

Whence the Coyotes?

John Durant, writing in the *Ford Times* has given a brief resume of the history of this breed. How the coyotes got into New York to furnish their share of the genes for this new predator remains something of a mystery. The Adirondack wilderness is hundreds of miles beyond the natural range of the coyote. Possibly pet coyotes may have escaped from captivity. The first coy-dogs appeared about fifteen years ago, and were mistaken for timber wolves, a species which disappeared from New York before 1900.

That the coy-dog might become a harmful predator is possible. He appears to be omnivorous with a preference for meat. He is said to somewhat resemble the fox in his pilferings around farm yards, but he is harder to trap than a fox, so the game wardens say. The claim that he is a sheep killer has, apparently, not been proven.

Perhaps the real role of the coy-dog is to maintain an ecological balance currently badly upset in this region. The late Aldo Leopold commented as follows concerning the sudden re-emergence of deer in the northeastern states: "In other instances we take credit for shaking down apples which were in all probability ecological windfalls. In the lake and northeast lumbering states pulping and fire accidentally created some scores of millions of acres of new second growth. At the proper stage we find these thickets full of deer. For this we naively thank the wisdom of our game laws."

Many of these thickets are becoming so heavily populated with deer as to create a serious problem for farmers in the cleared land in the valleys. Over much of this range, the deer populations are reaching a density which may soon menace their own survival—as happened in the Kaibab plateau some years ago. Perhaps the role of the coy-dog is to act as a positive check in the Malthusian sense on further multiplication of deer.

If that does happen, it will constitute an instance of an "ecological windfall" created *de novo*, and quite beyond the area of conservation action.

To students of evolution, the emergence of this new type in a region that has heretofore been definately outside the range of one of the parent species has elements of considerable interest. Will time and the selective forces acting on dog and coyote genomes, evolve a new species peculiarly adapted to the wild mountain areas of the northeastern states?

Geneticists will watch for futher news of the coy-dog with considerable interest.—R. C.

TAUPE AND PINK-EYE MICE

Figure 6

A and *A'* are dorsal and ventral views, respectively of non-agouti pink-eye. *B* and *B'* are non-agouti taupe. The black pelage of the back, normal for C57 mice has been replaced by gray. Taupe females have a very high fetal mortality and have never succeeded in raising their own young.

THE TAUPE MOUSE

A New Coat Color Mutation

JUDITH H. FIELDER*

IN December, 1948, six young were born to a pedigreed pair of C57 black subline 10 mice, the litter representing the 46th generation of continuous brother × sister matings. Upon growth of the coat it was observed that four animals were black, as expected, while two were distinctly gray in appearance.

Description

This new coat color, given the name "taupe" (symbol *tp*), resembles ruby, but differs in that the belly fur is lighter, with distinct yellow at the margins, and the eye color is not affected.

It was observed that although many homozygous taupe females became pregnant, none successfully raised her litters. One hundred and nineteen pregnancies were recorded among 64 such females, but only 73 litters were found, many of them dead. A number of young, of both those born alive and those born dead, were observed to be unusually small, though other animals in the same litters were of normal size.

Several interesting observations were made on these 64 females and their litters. Five, apparently in the late stages of pregnancy, were found to be having difficulty in labor. These were killed and the uteri opened. In four cases dead fetuses were found, a partially resorbed embryo apparently blocking the passage in one instance. The uterus of the fifth female contained resorbed embryos. In each case it was the female's first pregnancy.

Three additional females were killed in the early stages of parturition with the hope of getting living young. Of a total of 15 fetuses, 11 were alive. Four fetuses, comprising the whole of one litter, were unusually small and died almost immediately. The remaining seven were provided with foster mothers but did not survive.

Five females were found dead within several days of pregnancy having been recorded, with dead young usually being found in the cage or protruding from the vagina. Another female which was recorded as being pregnant was found 13 days later with the vagina extruded and no trace of the litter, which apparently had been produced with difficulty.

Examination of adult taupe females revealed a lack of nipple development, with most or all of the nipples being difficult to detect. Subsequent microscopic study of the mammary glands showed normal development of the ducts and alveoli. Histological examination of the ovaries of four adult homozygous taupe females showed that they contained normal follicles and corpora lutea.

While no taupe females have reared their own young, a number of young born alive to taupe females have been reared successfully by foster mothers.

Genetics

Preliminary tests indicated that the mutant, a simple recessive, is distinct from dilution, leaden and chinchilla and is not an allele of ruby. Crosses with pink-eye gave normal appearing F_1 mice. Subsequent testing has shown the taupe gene to be on the first chromosome and linked with pink-eye, although its precise location has not yet been determined.

Out of 195 F_2 from crosses of pink-eye × taupe, there were no crossovers. The results are summarized in Table I.

Of the 59 mice classified as $p+$, nine females and one male produced sufficient offspring in test matings to homozygous taupes to prove that they were not carrying taupe.

In the absence of linkage between taupe and pink-eye, one out of four pink-eyed mice should be homozygous taupe ($tptp$) and two out of four heterozygous for taupe ($Tptp$). Hence the chance that ten pp mice would all be homozygous non-taupe ($TpTp$) is $(\frac{1}{4})^{10}$ or one in 1,048,576. This removes any possibility that the apparent linkage is due to such a close resemblance between pink-eyed taupe and pink-eyed non-taupe mice that the former were not recognized as a distinct class.

In the absence of proved crossing over, taupe must be regarded as possibly an allele

TABLE I. Linkage of taupe with pink-eye.

Matings	++	p+	+tp	ptp
$\frac{p+}{+tp} \times \frac{p+}{+tp}$	91	59	45	0

*Jackson Memorial Laboratory, Bar Harbor, Maine. This investigation was supported in part by a research grant from the National Cancer Institute, of the National Institutes of Health, Public Health Service. The generous assistance of Dr. George D. Snell in the compilation of this material, and of Drs. E. Fekete and F. Richardson in the examination of histological preparations of ovaries and mammary glands is gratefully acknowledged.

of pink-eye. The normal appearance of the heterozygote, however, argues against this conclusion.

The gene is now being maintained by mating heterozygous females and homozygous males.

Summary

Taupe (*tp*), a new recessive coat color gene, lightens the coat and is responsible in homozygous females for the inability to nurse the young due to lack of nipple development. In linkage tests no crossovers with pink-eye have been found. The pink-eye taupe heterozygote is normal in appearance. It remains to be determined whether the genes are allelic or at separate loci.

(Received November 7, 1951)

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THE EUMELANIN HORSE: BLACK OR BROWN?

To the Editor:

In a recent issue of the Journal of Heredity (vol. 42, page 297) Prof. Castle reviewed my book, *"A los Colores del Caballo"* with a great depth of understanding and wealth of detail: more than enough to make the author profoundly thankful and also very appreciative of how the reviewer surmounted the difficulty of a foreign language. Owing probably to this handicap, the following three points of the book were not, I feel, truly presented in the review:

1. The postulated mutation from A to a^t is not supposed to give rise to the phenotype of the ordinary brown horse, but to that of the "seal brown." This was accurately described by Gremmel as a black with the so-called "light points," a phenotype in which I find close parallelism to the "black and tan" of other mammals. I have seen horses and of course many asses in which the distribution and sharp contrast of the "light" areas was exactly the same as that of a "black and tan" rabbit.

Castle states there are no true-breeding browns, but the fact has never been proven for seal browns. In any case, as I pointed out in this Journal in 1940, horsemen often discriminate very imperfectly among the terms of the series bay—brown—seal brown—black, and therefore any appreciation of their heredity based solely on Stud-Book statistics is unreliable. Only the experimental approach could solve the question.

2. According to the reviewer, the author "regards all eumelanin of horses as black, and explains the difference between bay and chestnut as due to a quantitative difference in black pigment. Others regard the difference as qualitative, the eumelanin in one case being black, in the other brown."

That does not define my attitude. As commented in the final paragraph of the same review I think it is "in the cards" the eventual apparition, in horses, of a b mutation, changing the black eumelanin to brown or rather chocolate. Indeed I went further, pointing out the possibility of a few bb horses actually existing, "lost" among the more numerous liver chestnuts—that I label ee as any other chestnut—because the latter's concentration of red pigment, going almost over the boundary in the so-called "black chestnuts" (one such was the famous *Domino*, as indicated by his offspring) would not make the horse's appearance, in some cases, very different from the chocolate.

Shades of the chestnut—from the lighter sorrels to the "black chestnuts" through the red and liver chestnuts—are difficult to sort out by means of factorial differences, multiple factors being apparently at work. The breeding results (as given by Salisbury, for instance) disprove the idea of the liver chestnut being just the aa chestnut—a hypothesis put forward by Sewall Wright in 1917 and accepted by Castle but not so in my book, although the reviewer's Table I is arranged as if I accepted it.

Concerning the difference between bay and chestnut, it is true I think of it in terms of E versus ee. But in my physiogenetical scheme it does not mean that E is conceived as a factor for "Extension of black"—Castle's own idea, suggested about rodents in 1909—but that e is taken to represent the "erythristic" mutant—a mutant found almost always in those mammals in which the intensity of pheomelanin in the wild (abouti) type reaches the red level of expression: to mention only one species, in the polecat.

3. The reviewer writes: "The genetic

(Continued on page 96)

WHISKER-EATING MICE

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IN his original description of whisker-eating, Hagedoorn[3] attributed the tonsorial hunger of some mice for the vibrissae of their cage-mates to a monogenic recessive operating through a nutritional deficiency. Since no breeding data were given in Hagedoorn's brief report, Grueneberg[2] refrained from expressing an opinion on the hereditary basis of this behavior trait but emphasized the possibility that mice might rub off their own whiskers because of the widespread occurrence of (temporarily) whiskerless animals in almost every stock kept by him, including several pure lines. Wire-mesh on cage lids and sharp edges on food hampers may sometimes be responsible for bald patches about the nose, but such accidental loss of vibrissae is probably much less frequent and less defined than the aesthetic depilation practiced by whisker-eaters.

The genetic and behavior observations reported here were gathered over the past year in our animal colony, which has a current annual production rate of about 34,000 mice of eight standard sibmated strains as well as hybrids of known genotype and the Webster-Swiss general purpose stock. Uniformity of mating schedules, balanced diet[6], bedding, cage population and other environmental factors facilitated a comparative survey for the incidence of whisker-eaters in relation to genotype, sex and age.

Procedures and Observations

The bilateral bald patches about the nose and orbital regions, characterizing the work of a whisker-eater, are pictured in Figure 7, which shows a C3H male with vibrissae and his three female objects of attention. The pattern is inflicted systematically, one side of the face and one orbit often being completely denuded before the other. This initial unilateral depilation was found either on the right side only or on the left side only in as many as five recently introduced cage mates of some whisker-eaters, and may indicate preferences in handedness.

Table I lists the various strains and genotypes in which whisker-eaters occurred, and the incidence among 1,962 adult mice. The trait came to expression in 40 out of 1,001 ♂♂ (3.99 percent ± 0.62), and 43 out of 961 ♀♀ (4.47 percent ± 0.66). This difference between the sexes is not significant.

The 83 whisker-eaters were discovered in a total of 68 cages, 51 of which were stock cages, each containing eight like-sexed individuals, and 17 breeder cages, each housing one male and one to three females with or without young. In 10 of the 17 matings the male was the whisker-eater, in seven the female was so inclined. The suckling young in such families—in one case 24 of them—were as neatly trimmed as the adults.

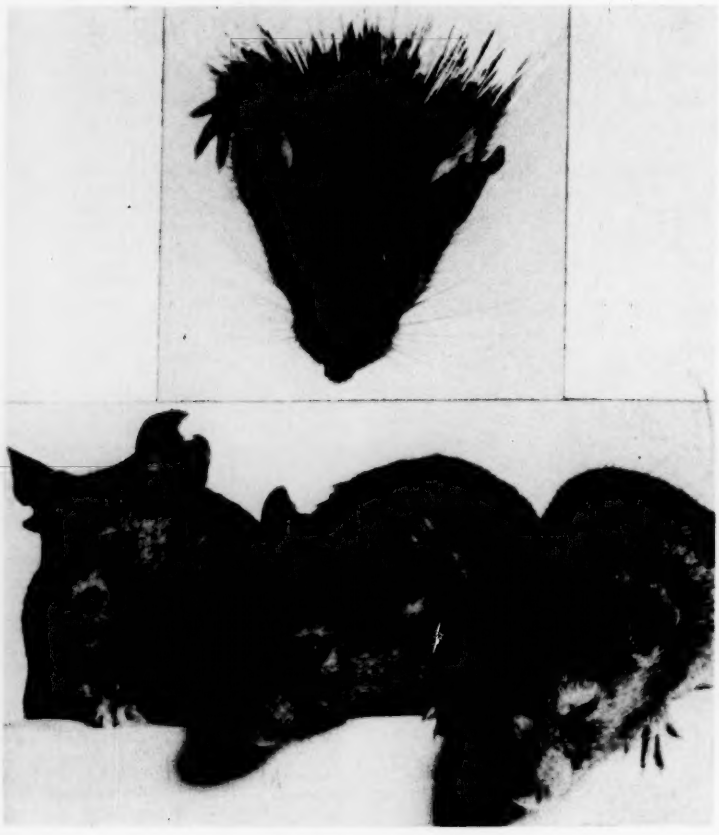
All the mice surveyed in Table I were fully mature or old animals, since the two youngest exponents of the habit ever found were between 1½ and two months old and since a comparison of incidence in three age groups (Table II) had shown a significant increase with age.

No whisker-eaters were seen among over 2,000 adult mice of the A, dba, C58 black, 129/Runner Jax, and Y/Heston strains, nor in 38 (C3H × A) F_1, 225 F_2 from matings between (C3H × A) F_1, and 135 F_2 from crosses between (C3H × dba) F_1. In backcrosses of both these F_1 types to C3H, whisker-eating occurred with an incidence of 1.14 percent and 2.89 percent, respectively (Table I). While the restriction of the trait to certain strains and the relatively uniform incidence in some genotypes involving C3H may have genetic implications, the hereditary mechanism is certainly not monofactorial, as is evident from the following backcross data.

Three daughters of a C3H ♂ whisker-eater whose performance had been repeatedly tested (Figure 7) were mated to their father. Sixty-five offspring from this backcross were periodically checked over a period from 3½ to 6½ months. On Hagedoorn's[3] postulate of a monogenic recessive, an incidence of 50 percent whisker-eaters would be expected among these mice, but none has appeared.

When Whisker-Eaters Meet

A few brief exploratory tests were aimed at the significance of whisker-eating in murine social structure. Four male and two female C3H whisker-eaters, all in characteristic full possession of vibrissae and of demonstrated depilatory



CLOSE SHAVE!

Figure 7

Above is shown a C3H whisker-eater complete with vibrissae. Below are his three cagemates, all clean-shaven on nose and orbits. A confirmed whisker-eater—and the tendency appears to be located in his genes—proved fully a match for a rat twice his size, removing all whiskers down to the skin in 24 hours. As yet cats seem to be safe from the inroads of these murine tonsorial artists.

skill, were matched as like-sexed pairs in three separate cages and their adjustment toward one another was observed daily for ten days. Immediately after introduction, the animals registered excitement manifested by fur-fluffing, labored breathing, and tail-twitching. In alternate advances, one paw was put firmly on the opponent's forehead; ears, face and neck were licked and vibrissae were nibbled. The passive mouse appeared cowed and retained a hunched pose—eyes tightly shut—for a few seconds after each attack; but signs of fear disappeared as the new experience became familiar. In all three cages dominance of one whisker-eater over the other could be diagnosed within 24 hours

from the skillfully denuded nose and orbits of the losers. Every winner retained a somewhat deteriorated mustache during nine days of further observation, after which the three winners and the three losers were separated into six individual cages and each was given two normal like-sexed cage mates, one of which was of the same strain (C3H), while the other was an A-strain albino. Similar companions were allotted to three control whisker-eaters not previously tried in competition. The results for these nine trios (Table III) are indicative of discouragement among the losers and suggest preference of pigmented C3H vibrissae over albino whiskers, but no pronounced aversion to the latter. The habit is apparently not limited by strain-specific taste, nor is it bound by species restrictions. An 83 g. male Wistar rat stoically tolerated the inroads of a 34 g. male C3H mouse on its luxuriant facial decor and was smoothly shaven without sign of a fight or subsequent fear.

Discussion

Aside from elusive temperamental characteristics, such as savageness, tameness and fighting[7], the only mouse behavior trait for which the inheritance has been elaborated is susceptibility to audiogenic seizures. For the latter phenomenon considerable strain differences have been reported by Hall[4] and Vicari[10]. As in the case of whisker-eater[3], the incidence of which also varies in different strains, the difference in response to sound was at first ascribed to a single pair of alleles[5]; but it was later found by Fuller, Easler, and Smith[1] to be a continuously distributed character of a quantitative nature. A similar multiple factor background cannot as yet be ruled out for whisker-eater, while single recessive factor control may certainly be discounted on the basis of our backcross data and the penetrance observed in F_1 hybrids between susceptible and non-susceptible genotypes.

Some evidence of maternal or litter-effects, the whisker-eating habit being more frequent among members of some litters than it ought to have been under random distribution, was obtained by Latyszewski and Falconer (personal communication, 1951). They also proved that hairs were eaten but not digested and tried various methods of "cure" in the hope of elucidating a dietary or hormonal mechanism. Feeding with chopped hair, keratin, cystein, or yeast had no effect. Castration and ovariectomy of either old or young mice neither discouraged the already established "vice" nor interfered with its onset.

Future work might advantageously be oriented toward the socio-psychology

TABLE I. Incidence of Whisker-Eating in Adult Mice of Various Genotypes

Genotype or Strain	Total Mice Examined	Whitsker-Eaters	Percent Incidence
C57 Black	461	46	9.98 ± 1.37
C3H + milk factor	347	11	3.17 ± 0.89
C3H — milk factor	162	5	3.02 ± 1.30
(C3H — × dba) F_1	46	3	6.52 ± 3.61
(C3H — × dba) F_1 × C3H —	138	4	2.89 ± 1.45
(C3H+ × A) F_1 × C3H+	176	2	1.14 ± 0.77
Webster Swiss	632	12	1.89 ± 0.54
Total	1962	83	4.23 ± 0.44

TABLE II. Influence of Age on Whisker-Eating in C57 Black Mice

Age Group	Age in Months	Total Mice Examined	Whisker-Eaters	Percent Whisker-Eaters	D/SE Ratio
I	4–6	221	6	2.71 ± 1.09	I and II 2.9
II	7–9	117	17	10.81 ± 2.98	II and III 1.7
III	10–12	129	23	17.83 ± 3.31	III and I 4.1

TABLE III. Decline in Performance of Defeated Whisker-Eaters and Preference for C3H over A-Vibrissae

C3H whisker-eaters scored in previous contest and controls	Number of days spent in dewhiskering two neutral cage mates	
	C3H	A
♀#1 (winner over ♀#2)	1	1
♂#3 (winner over ♂#4)	1	1
♂#5 (winner over ♂#6)	1	25 (incomplete)
♀#2 (loser to ♀#1)	25 (incomplete)	not dewhiskered at 25
♂#4 (loser to ♂#3)	3	11–11
♂#6 (loser to ♂#5)	25 (left side only)	25 (left side only)
♀#7 control	2	1
♀#8 control	11–13	15–17
♂#9 control	2	11–13

and psycho-genetics of whisker-eating. Seward[8,9] has concluded from his investigation of the grooming drive in rats (which here manifests itself as licking and chewing, but not removal of hair) that the desire to groom is closely allied with the desire to be groomed. Thus, if more than one whisker-eating mouse arises among the population in a cage the competition for active practice of this exaggerated mode of grooming will soon result in dominance of the most efficient practitioner and in subordination of less efficient and purely passive animals into a closed circle of mutual enjoyment. The exploratory experiments reported here suggest the potential usefulness of this trait in studies of handedness, of competitive adjustments and of grooming interrelationships as clues to an otherwise cryptic dominance hierarchy.

Summary

Whisker-eating mice, whose performance followed a precise working pattern, occurred in several heterozygous genotypes and in two inbred strains, but not in five other pure lines —all on the same diet. Inheritance of the character is not monofactorial, as claimed in the literature. Multiple factors may be involved. Dependence of manifestation on age was established. Exploratory experiments were directed at an interpretation of the trait in terms of the grooming drive, competitive adjustments, and social hierarchy.

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SEX MORPHOLOGY AND MECHANICS

THE second edition of the "Organism and Sexuality," by the venerable Maurice Caullery,* summarizes an immense amount of observational and experimental information on primitive sexuality, parthenogenesis, hermaphroditism, secondary sex characters, sex hormones, chromosomal and environmental sex determination, genetic and experimental intersexuality, gynandromorphism, and related topics. The situation found in various subdivisions of the animal, and often also of the plant kingdom, is reviewed in a systematic fashion. Unfortunately, the role of the sexuality in evolution is not discussed at all; without such a discussion the biological meaning of the phenomena described in the book is not apparent to the reader; the great variety of methods of reproduction found in the living world becomes a mere collection of curiosities.

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*M. Caullery. *Organisme et Sexualité. Encyclopedie Scientifique.* Second Edition. G. Doin, Paris. 490 pp., 131 figs. 1951. (2300 francs).

INHERITANCE OF NATURAL AND INDUCED MUTATIONS IN CALORO RICE

And Observations On Sterile Caloro Types

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THE Caloro rice variety was selected in 1913 at the Biggs (Calif.) Rice Station from Early Wataribune, and was released to growers in 1921. It is a midseason, partly-awned, short-grain variety of Japanese type. Caloro heads and matures uniformly and produces relatively high yields of reasonably good milling quality rice (See Figure 8*A*). Caloro soon became the main commercial variety in California and for many years has been grown on 80 percent or more of the annual rice acreage in that state. For many years Caloro also has been grown in all cultural, irrigation, and fertilizer experiments conducted at the Biggs Rice Station. Close observation of the growth habit and other characteristics of Caloro, at the station and on commercial fields, has made it possible to detect mutations and other off-type plants readily. Many of these were collected, grown, and crossed with normal Caloro. The F_1 plants were normal in appearance and their mode of inheritance is shown in Table I.

Natural Mutations

Some of the mutant forms are described as follows:

Compact Variant (Figure 8*B*)—Plants of this type have short stiff, relatively thick culms, erect, compact spike-like panicles, and plumper seed than that of normal Caloro. The average height of normal F_2 segregates was 40, and variant segregates 35 inches.

Physiological Disease I (Figure 8*C*)—This disease affects and discolors plant leaves, culms, and panicle branches. The height and vigor of affected plants is reduced, particularly the recessive type. The average height of F_2 normal and intermediate segregates was 37 inches, and of the recessive diseased type 23 inches.

Physiological Disease II (Figure 8*D*)—In affected plants, the disease appears on the leaves, culms, necks, branches of the panicles; and even the hulls turn brownish as the plants approach maturity. Diseased plants are somewhat shorter, and lodge more easily than those of normal Caloro.

Hull spots (Figure 8*E*)—In this mutation the plants have sesame-shaped thin-textured, colorless hull spots. The spots are devoid of color at flowering and for a short time thereafter. The spots, however, usually become discolord before maturity. Both the growth and the seed of affected plants appear to be normal in size and vigor.

Short Internodes (Figure 8*F*)—In this non-heading (Sathi) type of mutation, the panicles are entirely enclosed by the boot leaves. This recessive type, however, sets some seeds cleistogamously even though the hulls are unable to open at flowering time, and the internodes are about one-fourth normal length. The intermediate heterozygous short internode plants have relatively short, partly exerted panicles and slightly smaller seed than that of normal Caloro. Seed of heterozygous plants give normal, intermediate, and non-heading plants in a ratio of 1:2:1.

TABLE I. The Segregation of Natural and Induced Mutations in Crosses With Normal Caloro.

Type of Mutation	*Number of F_2 Plants* Normal	Segregating	Mutant	Total	Ratio
Natural					
(a) Compact variant × Caloro	214	—	74	288	3:1
(b) Physiological Disease × Caloro	188	362*	179†	729	1:2:1
(c) Physiological Disease × Caloro	256	—	85	341	3:1
(d) Hull spots × Caloro	185	—	58	243	3:1
(e) Short internode (seg.)	131	241‡	120§	494	1:2:1
(f) Rolled leaf × Caloro	497	—	159	656	3:1
(g) Neck leaf × Caloro	266	—	82	348	3:1
(h) Coarse culm × Caloro	311	—	92	403	3:1
(i) Haploid ♀ × Caloro	255	—	83	338	3:1
Induced (X-ray)					
Partly Sterile Compact × Caloro	218	—	72	290	3:1
Short small seed × Caloro	208	—	84	292	3:1

*Intermediate disease
†Recessive disease type
‡Intermediate internode
§Very short recessive internode

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RICE MUTATIONS

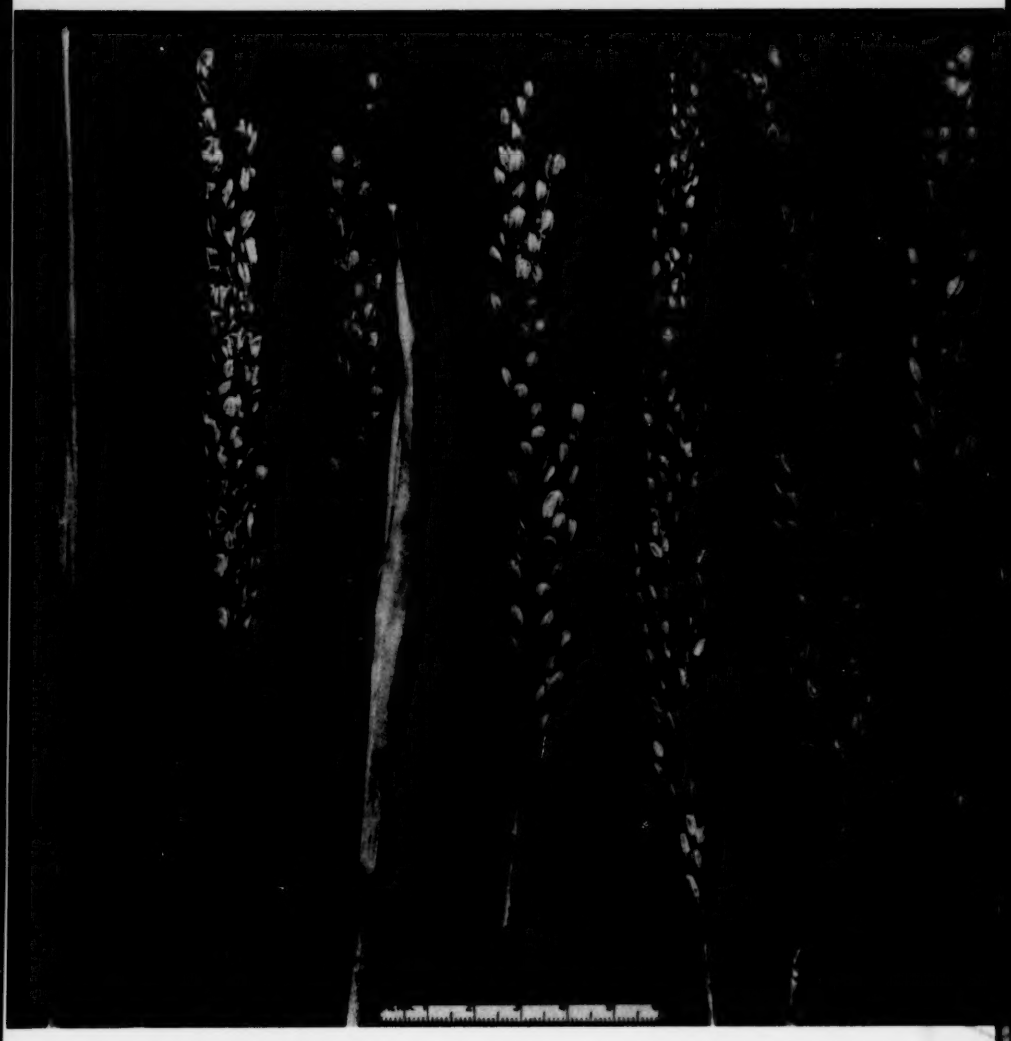
Figure 8

A head of normal Caloro rice is shown at *A*. The mutants are discussed in detail in the text. All the mutations occurred spontaneously except mutants *K* and *L* at the extreme right. These were induced by treatment with X-rays.

Rolled Leaf (Figure 8*C*)—In this mutation, the comparatively short, partly sterile plants have rather narrow leaves that fold inward. The sterility appears to be due to malformed florets. The lemma and palea (hulls) often fail to interlock normally after flowering, thus leaving the upper part of the kernel exposed. The seed produced is smaller than that of Caloro.

Neck Leaf (Figure 8*H*)—Normally in rice, leaves do not develop above the flag-leaf, though occasionally one or two small leaves may develop at the base of a panicle, but such variations usually are not hereditary. A recessive hereditary neck-leaf mutation was found, however, in Caloro at Biggs. Except for the presence of neck-leaves, the plants were of normal appearance and vigor.

Coarse Culms (Figure 8*I*)—The culms and panicles of this mutation are somewhat more erect than those of normal Caloro. Both of



these characters were apparently recessive to normal. However, there was a deficiency of the recessive class, owing apparently to difficulty in correctly classifying the mutant type.

Haploid 3—In this mutation, the small sterile plants have short panicles and small florets of variable size. Thus the florets are unlike those of typical haploid plants. The mutant plant was pulled, sent to Washington, and transplanted in a greenhouse. The following spring, the potted plant was placed in a screened inclosure outside where it produced two small seeds. These seeds were sown in a greenhouse, and produced partly sterile normal-like plants and seed. Seed from one of these plants, sown at Biggs, California the following year, produced only plants similar to those of the original haploid 3 type. However, two small seeds were found on separate plants. In the greenhouse, these seeds also produced partly sterile normal-like plants and seed. The fact that seed from haploid 3 plants sown in a greenhouse in late fall produced partly sterile normal-like plants and seed in the following spring, whereas it produced only haploid 3 type plants when sown in the field in the spring at Biggs, California, clearly indicates that the genetic factor responsible for the haploid 3 mutation is markedly affected by environmental conditions. It is not known whether the effect resulted from differences in sunlight intensity,

temperatures, day length, or to a combination of these factors. One of the partly sterile greenhouse plants was crossed with normal Caloro. The F_1 plant was normal in growth and seed type, and the F_2 population, grown at Biggs the following year, segregated in a ratio of 3 normal to 1 haploid 3 type plants.

Induced Mutations

Two mutations were selected at Crowley, Louisiana, from plants produced by X-rayed Caloro seed.

Compact Sterile (Figure 8*I*)—This partly sterile mutation has compact panicles, somewhat deformed seeds, with long outer glumes or an additional glume which often is rather long.

Short small seed (Figure 8*K*)—In this mutation the plants are shorter and produce smaller seed than those of normal Caloro. Both of these mutations, from X-rayed seed, were recessive to normal Caloro.

Sterility in Caloro

A number of partly sterile Caloro plants were selected at Biggs in 1938 and 1939. These consisted of (1) partly sterile normal-like plants, (2) plants with twisted florets and somewhat smaller seed than Caloro, (3) plants with somewhat compact panicles, and (4) a few short aneuploids, plants with $2N + 1$ and $2N + 1 + 1$ chromosomes.

Forty-nine of these selections were grown in spaced rows at Biggs, California, during the three-year period, 1941-1943 and 46 lines were grown at Beaumont, Texas, from 1940 to 1942. The amount of sterility in the lines grown was determined by counting the number of seeds and sterile florets on five panicles of each line collected at random.

Normal Caloro had an average of 4.8 percent of sterility at Biggs and 7.5 percent at Beaumont. The three-year average sterility of the lines grown ranged from 4.4 to 94.4 percent at Biggs and from 7.8 to 92.2 percent at Beaumont. At Biggs the average weighted sterility of all lines was 51.5 percent and at Beaumont 36.5 percent, respectively. These partly sterile types were influenced by both heredity and environment.

Sterility in Tetraploid Caloro

In 1938 a tetraploid plant was found in the Caloro variety at Biggs, Calif., and the following year a mosaic tetraploid also was found in Caloro. These tetraploid plants are shorter, do not tiller so well, have coarser stems and leaves, larger florets and seed, mature somewhat later, and are much more sterile than normal Caloro plants. Parthenocarpic ovary development (water bugs) is much more common in tetraploid than in normal Caloro plants grown under like conditions[1].

These observations are in agreement with the findings of Morinaga and Fukushima[2]. They reported that (a) the tetraploid plant is somewhat stunted, produces fewer productive tillers, has coarser leaves, fewer spikelets per panicle, larger seed, and the awns better developed than that of the original diploids. The pollen-grains also were larger than those of the diploid floret, and about 50 percent of the florets were empty and abnormal in shape. The degree of sterility varied in tetraploids of different origin and ranged from 62 to 94 percent. A tetraploid that had been grown for nearly 15 years, before its chromosome number was determined had about 70 percent of sterility.

In 1940, seed of a tetraploid (p. 15-1, pl. 3) was space-sown in two rows at Biggs, Calif. In one row of 20 plants, the degree of sterility ranged from 15.3 to 94.2 percent, with an average of 31.6 percent. In the other row, of 21 plants, the range of sterility varied from 19.0 to 58.3 percent, with an average of 30.2 percent. In the same year, diploid Caloro, grown under like conditions, had only 2.7 percent of sterility.

In succeeding years an effort was made to select more fertile tetraploid lines. Seed of the four most fertile plants in group 1 and of

TABLE II. Sterility in tetraploid Caloro rice at Biggs, California.

Plant No. from Tetraploid P. 15-1, pl. 3	Year and Sterility in Percent										5-year (1940-44) Average
	1940*	1941 Range	1941 Av.	1942 Range	1942 Av.	1943 Range	1943 Av.	1944 Range	1944 Av.		
Group 1											
Pl. 12	15.3	36.1-100	60.5	29.9-98.9	53.2	21.5-95.3	56.5	37.0-100	53.1		47.7
Pl. 15	17.3	34.2-100	54.2	36.2-100	58.3	52.1-99.0	71.0	42.2-100	56.6		51.5
Pl. 1	27.0	33.5-100	49.9	36.8-100	52.6	49.7-100	68.5	36.7-100	58.5		51.3
Pl. 2	29.1	33.8-100	52.4	45.1-100	62.9	56.9-100	78.5	35.0-100	62.4		57.1
Group 2											
Pl. 10	51.7	33.6-99.7	54.6	71.5-78.4	75.7	48.3-97.3	67.1	39.3-100	59.5		62.1
Pl. 19	56.9	36.8-100	52.8	41.7-100	60.1	51.7-98.7	70.6	39.7-100	63.2		60.7
Pl. 20	92.0	88.7-100	88.7	48.1-100	69.9	38.7-93.6	69.8	44.0-100	66.2		77.3
Pl. 3	94.2	39.4-99.2	54.9	53.2-100	69.4	26.7-100	68.1	40.1-100	61.9		69.7
P. 15-1, Pl. 3	31.6	33.6-100	58.5	48.1-100	69.9	29.2-98.0	45.4	33.5-94.9	57.0		52.5
Cal. 19	75.9			53.2-100	69.4	63.9-98.7	64.8	53.2-100	71.4		70.4
Normal Caloro	2.7		10.1		7.7		5.4		5.4		6.3

*Range 15.3-94.2 average 31.6 percent for Tetraploid (P. 15-1, pl. 3)

four much less fertile plants in group 2 in 1940 were space-sown in 1941. In succeeding years, seed of the most fertile plants in group 1, and seed of plants having 50 percent or more of sterility in group 2 were space-sown.

The sterility observed during five years of testing is shown in Table II. In group 1, the 5-year (1940-44) average sterility ranged from 47.7 to 57.1 percent, and in group 2 from 60.7 to 77.3 percent. In the same period, the sterility of the unselected parent (P. 15-1, pl. 3) was 52.5 percent, or essentially the same as that of the original plants grown in group 1, but considerably less than for the plants of group 2. In normal Caloro, sterility ranged from 2.7 percent in 1940 to 10.1 percent in 1941, with an average of 6.3 percent. In both groups there was a wide range in sterility each year. None of the plants in group 1 was as fertile in succeeding years as the original plants were in 1940, but in group 2, except for No. 10 (in 1942) some plants had less sterility each year than the original plants. Selection for four years of the most fertile lines in group 1 failed to reduce sterility materially as compared with that of the original tetraploid plants; whereas two lines in group 2, having over 50 percent of sterility, tended to increase in average sterility and the other two lines to decrease in sterility during the period grown.

Summary

Natural mutations in Caloro including compact variant, two types of physiological disease, hull spots, short internodes, rolled leaf, neck leaf, coarse culms and haploid 3 were all simple recessives. Other mutations found include albino and yellow lethal seedlings, white and yellow striped plants, male sterile and other sterile types. The following chromosome aberrant forms also were isolated from Caloro at Biggs, Calif., haploid, triploid, tetraploid, and aneuploid ($2N + 1$ and $2N + 1 + 1$) types.

In a four-year period, it was not possible to improve the fertility of tetraploid lines by selection.

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THERMODYNAMICS AND EVOLUTION

Dr. Harold F. Blum* has undertaken a formidable task: investigation of the physico-chemical bases of organic evolution. It will be generally agreed that such an investigation is timely. Since the living world is the outcome of evolutionary development, the structures, functions, and ways of life of organisms have arisen as a result of a long historical process. Evolution is, accordingly, the unifying idea which can tie together the biological sciences. A trend towards emergence of an evolutionary biology has developed particularly within the last two decades. Genetics, cytology, systematics, paleontology, ecology, comparative morphology and embryology have all participated in this trend, but the great fields of physiology and biochemistry have so far remained preoccupied with their own basic problems. Books have been published entitled "Physiological Evolution" and "Biochemical Evolution," but they showed little awareness of the modern evolutionary thought. This is certainly not the case with Dr. Blum, who states his approach very succinctly thus: "To what extent has the course of evolution been determined by strictly physical factors that have permitted no exercise of natural selection, and to what extent have the former set the limits within which the latter might act? Can the two things be clearly separated, and what errors of interpretation may be made if we are unable to do so?"

Unfortunately, the book does not give answers to these very clearly stated questions, beyond the rather commonplace consideration that the second law of thermodynamics (the "Time's Arrow") applies to living systems. The author discusses a wide range of subjects, all the way from the origin of the Earth and the planetary system, of the continents and the oceans, the fitness of physical and chemical environments for life, the energetics and kinetics of biological systems, the self reproduction of genes and chemical molecules, the origin of life (where he has a hypothesis of his own to propose), and to the irreversibility of evolutionary changes. The author is certainly unafraid of free speculation and of uncharted spaces of imaginative theorizing. Interesting and suggestive ideas are liberally scattered in many of the chapters. But a book that would integrate the fields of physiological and chemical biology with the modern ideas about the organic evolution still remains to be written.

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*Harold F. Blum, *Time's Arrow and Evolution*. 222 pp., 30 figs. Princeton University Press. 1951. ($4.00).

GENETIC THEORY IN THE PYTHAGOREAN SCHOOL

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THERE are two reasons for a contemporary geneticist to be interested in Pythagorean speculation on heredity. The first is that scientific genetics originated with this school. The second is that looking at earlier attempts to apply mathematics to complex phenomena may suggest fruitful approaches to a contemporary situation in which new discoveries already promise to upset the elegant simplicity of an atomistic genetic theory. The Pythagoreans, committed to field rather than atomic types of scientific explanation, produced what seems a very different type of theory from our current ones. Looking at such an alternative approach to his subject-matter may have for the geneticist some of the values that studying non-Euclidean geometries has for the mathematician. Thus a summary of the earliest history of genetics may not only remind us of the honor which the Pythagorean scientists deserve, but may suggest new methods of approach to the building of theories. References are given for the reader interested in verifying and evaluating the sources involved, but this discussion will be a summary of the theory as reconstructed without digressions into technical niceties of that reconstruction.

Beginning of Genetics

The Pythagorean research program of applying mathematics to phenomena[1] (begun ca. 529 B.C.[2]) led them to try to find some quantitative key to the interesting phenomena of heredity. This attempt was the beginning of genetics as a science. They seem to have begun with the most interesting, but most complex, phenomena; the hereditary component in complex traits of human "character."[3] This beginning may have been influenced by the theories of the medical school of Crotona, which seems to have regarded the patient as a single psychophysical whole. Although this emphasis did not preclude study of anatomy and practice of dissection, it did direct attention to the interconnection of parts of an organism, and away from theories treating simple traits separately.[4]

But evidently a complex trait like "honesty" or "courage" is not purely the result of heredity; it must be thought of as a hereditary capacity, elicited by the environment.[5] The only way to measure such capacities is by observing their realization.[6] The key postulate is that if the capacity is not present, no environment can elicit it, though if it is, the environment can always frustrate its realization. Further generalization from their observations led to a second basic concept of this theory: in respect to any trait, the maximum development of a child will be about the average of the capacities of his parents. Third, it seemed clear that human character traits varied through a continuous range, requiring a geometrical, not an arithmetical, type of quantitative representation. The notion of the inherited average as (approximate) maximum was probably supplemented by a physiological theory in which the "seed" was treated as derived from and reflecting the entire parental organism, and the interaction of seeds as a kind of fusion creating a field of potencies.[7] We might note that of the possible notions of relation of parental seeds, the Pythagoreans tried the simplest, that of addition; in later speculation, more complex suggestions were offered; Empedocles introduced a mechanism of "dominance," Plato seems to have seen the need for some provision for "emergence," Aristotle uses terms that suggest the possibility of the two preceding types of relation plus something like "mutation."

The "Genetic Triangle"

Some mathematical device was needed to treat the relations of such "capacities." Apparently, what was done was to equate genetic "potencies" with recently discovered mathematical objects also called "potencies"; these were irrational lines, the squares (or higher powers) of which were rational. The mathematical "root" was not conceived as an actual magnitude (for a "magnitude" was defined as an integral number of units), but as an entity that had the power, when it "grew" into a

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square, of becoming rational and commensurable with magnitudes. Consequently, the symbol chosen by the Pythagoreans as the key genetic diagram was a right triangle, one side representing each parent, the hypotenuse their offspring, and the inheritance of a given capacity by the child being determined by its intensity in the parents.[8,9] In this way, inheritance could be treated as relevant to capacities for complex traits, without being directly causative of the traits in question.

To construct an analogue to this approach today, we would have to use different mathematical tools. If an individual's capacities at a given time were represented by a matrix showing the different probabilities of the possible directions and degrees of their realization, treating the present state as a kind of superimposition of these potential futures, the result would be a modern counterpart of the Pythagorean scheme. And the ontological status of these superimposed "present possible futures" would have just the equivocal character which irrational roots were originally defined as possessing.

Actually, the key genetic symbol of the Pythagoreans was not simply any right triangle, but the right triangle with sides of 3, 4, and 5. This symbol embodies several features which we find in their theory. The use of a geometrical figure recognizes the continuous range of traits studied.[10] The selection of 3 and 4 (following the notion of odd numbers as masculine, and even as feminine) reflects the qualitative difference between the parents. (It need not specify any quantitative differences between them.[11]) The use of a right-triangle gives the formula by which mathematical "potencies" may be compared in terms of the "magnitudes" into which they develop; the "Theorem of Pythagoras" being the basic tool for study and construction of irrationals.[12]

Inevitable Deterioration

One defect in this theory is its inability to account for either-or traits, or for inheritance which closely resembles one parental line but differs widely from the other.[13] A second defect, if scales of traits are constructed as Plato's are in the *Phaedrus* and *Republic*, with the best development at the top of the scale, set equal to 1, is the rapid deterioration in human aptitude which the theory predicts.[14] With scales set up in this way, whether the *average* or the *square root of the sum of squares* of parental potencies is used to calculate inherited endowment, the theory predicts an inordinate rapidity of human deterioration. Using the second calculation (which follows the theorem of Pythagoras more exactly) one would expect about one-seventh of each generation to be more degenerate than any of their ancestors, and four generations to eliminate all persons of a ton level of ability ranked as 1 on the initial scale.[15]

Of these two difficulties, the second seems to have awaited Plato's invention of eugenics for its discovery; the former, particularly in reference to the sex of offspring, was early recognized and accounts, probably, for suggestions that the determining factor here is not hereditary.[16]

This early venture in genetics has been described both for its interest as an important point in the history of science, and for its value as a very different way of building a theory to explain genetic phenomena. Although the theory seems to us over-ambitious in beginning with complex traits, naive in assuming that simple arithmetical relations (such as a sum or average) would describe the laws operative, and yet probably hard to apply because of its mathematical complexity, it served as the starting point of subsequent Greek speculation, and provided an incentive for further observation and quantification of phenomena. This line of thought strongly influenced Plato, who, in the awkward position of being unable either to accept the atomistic Hippocratic or Empedoclean theory, or to account for known phenomena by the earlier Pythagorean one, was in much the sort of dilemma toward which geneticists seem to be moving today.[17]

References

(1) For Pythagorean science, see J. Burnet, *Early Greek Philosophy*, 5th edn., and the brief summary in N. P. Stallknecht and R. S. Brumbaugh, *The Spirit of Western Philosophy*, Chap. I.

(2) 529 B.C. is the traditional date of Pythagoras' arrival at Crotona.

(3) Greek literature shows an interest in such inheritance of "character" from Homer on. The interest is natural, and assuming it as a starting-point gives coherence to the Pythagorean doctrines.

(4) The stress on the whole organism is characteristic of such later works as Empedocles' poem and Plato's *Timaeus*, that derive from it; whether it was pre-Pythagorean or an idea taken over from the Pythagoreans, the effect on their genetic theory would be the same.

(5) Or, of course, no hereditary factor may be involved in such traits at all. Plato's "case-studies" in *Republic* VIII-IX show how clearly he recognized the force of environment in forming personality, though there is no telling how far the earlier thinkers (prior to Empedocles) agreed.

(6) Our own measures of "aptitude" are always *performance* tests, and by definition there is no other way for aptitudes to be measured. The "postulates" are treated in (8), below.

(7) The notion of fusion of seeds, which appears in Plato, seems taken by him from Sicilian medicine, from a tradition different from that of Empedocles (in the *Timaeus*, Plato generates "seeds" from the "marrow"

which "coordinates" the parts of an organism).

(8) The basic source here is Plato's passage, *Republic* 546A ff. That passage is about genetics, and in the middle of it appears a 3-4-5 triangle which all scholars identify as a Pythagorean genetic symbol. Some of the extraordinary problems raised by the passage can be found discussed in J. Adam, *Republic*, II, Appendixes to Book VIII. I have assumed that the part of this passage subsequent to the triangle is an elaboration of the Pythagorean diagram, on the basis of Aristotle's comment (*Politics* 1316a) that the triangle is the "principle" explaining all the rest of the construction. I have assumed the first phrase to be Pythagorean in essence, because the triangular figure is grammatically represented as derived from it. If this is right, it may help explain why the effort expended on explaining this text by reference to *Platonic* theories has not produced any generally accepted complete interpretation; it may also explain why Plato presents this theory as a "story," as though not willing to take it entirely literally.

(9) The status of the irrational is discussed in Heath, *History of Greek Mathematics*, I. Plato's passage begins with a derivation of the number governing human generation from the "increase of roots to their squares." Aristotle's reference, *Metaphysics* 1091a, to Pythagoreans who talk about numbers as "generated from seed" preserves a comparable metaphor. The contrast of "roots" and "magnitudes" is made explicitly in Plato, *Theaetetus* 146a. Just before passing on to the triangle in the key *Republic* passage cited, Plato seems to complete his metaphor of "roots growing" with the statement that these increases "reaching three stages with four termini, make everything comparable and in ratio." The Hellenistic commentators believed that it was the fact of its being a right triangle that was the central point of the genetic symbol (see Heath, *Euclid*, I, 2nd edn., "Popular Names for Euclidean Propositions, I. 47"). That would point to the rule that inheritance equals the square root of the sums of the squares of parental endowment, rather than the simpler rule of the average which seems to apply in Plato.

(10) The notion that traits occur in multiples of some elementary quantity, not varying continuously, did not appear until Greek Physics had developed theories of "elements" which were definite quanta.

(11) Aristotle, *Metaphysics* 986a, quotes a Pythagorean table of opposites in which odd numbers are masculine, even numbers are feminine. Plato, *Timaeus* 35 ff., uses series of odd and even numbers to represent qualitative distinctions, and this seems to follow a Pythagorean tradition (see, on this point, A. E. Taylor, *A Commentary on Plato's Timaeus*, notes on 35 ff.).

(12) In spite of uncertainties of interpretation, one is tempted to see evidence that the "sum of squares" rule was used by the Pythagoreans in the triple repetition of an operation of "squaring" and in the reference to "rational diagonals" (nearest integral approximations to the values of roots) in Plato's passage.

(13) See (16), below.

(14) Plato's "scales" are those established by himself in *Phaedrus* 249 and *Republic* 587D. In the Pythagorean table Aristotle quotes, cited above, good and one, evil and many are grouped together, another indication that the best development of a trait would be ranked as 1 and put at the top of any ordinal scale set up by a Pythagorean. Plato himself changed this procedure, at least in his later dialogues, and cites evidence (at 424A) that contradicts it in the *Republic*.

(15) If we take a "generation" of statesmen as about twenty years, this theory would predict a radical change, for the worse, every 80 years. There seems no way of telling how far the Pythagoreans had worked out the social implications of their theory; it was just such implications which made genetics interesting to Plato.

(16) The solution adopted seems to have been to regard sex (and presumably all other either-or traits) as due to environmental factors, hence not hereditary at all. Parmenides and Empedocles both agreed to this suggestion.

(17) In the *Symposium*, Plato makes fun of the Hippocratic theory; in the *Republic* and *Timaeus* he shows his admiration for the elegance of Pythagorean mathematical science, but cites phenomena which their theory will not explain.

THE GENETIC ASPECTS OF MENTAL DISORDERS IN THE AGING

Comparative Twin Data on the Involutional and Senile Periods of Life*

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ALTHOUGH the effect of heredity as a determinant of variable adjustive potentialities is essential in every sector of the normal life span, its crucial testing stage is not reached before the *senium*. The biological integration of the human life cycle is apt to fall short of its real fulfilment without a patinated period of reflection upon the lasting value of man's creative and sentimental experiences. On this basis, the later years of life can be serene and rewarding only if they remain free of conflict and tension.

Unfortunately, senescence is often clouded by severe emotional disturbances, many of which have their roots in the early years of life before and after adolescence. In other words, the majority of mental disorders in later life are believed to originate from gradually accruing imperfections in basic adaptive capacities.[11] It is evident, therefore, that an understanding of the geriatric problems of psychiatry requires information about the *genetic* aspects of aging and old age. For comprehensive investigations of this kind, longitudinal twin studies have proved to be particularly useful.[7]

Generally speaking, the effect of genetic elements in relation to maladjustment in later life may express itself in three different ways:

1. The first possibility is that of genetic factors being responsible for the *pre-senescent* occurrence of disorders, physical or mental, which may lower adaptability to the impact of those morphological changes associated with advancing biological age. It is regrettable that present knowledge about the nature and range of protective and compensatory mechanisms, which qualify the constitutional capacity for adaptation to old age, is still very limited.

2. The second possibility is that of heredity being a major determining factor with respect to the normal average ability of man to survive up to and through the period of senescence. Obviously, a person must be sufficiently *longevous* to be capable of developing any type of senile disorder. It is also certain that the tendency to break down with the symptomatology of a senile psychosis presupposes the capacity for living through the earlier years of maturity without succumbing to other types of psychosis.

Variation in the Life-Span

Conclusive evidence of variable biological survival values in the absence of extraneous life-shortening adversity has been obtained with animal experiments,[6] with statistical family studies disclosing a positive correlation between the life spans of parents and their children,[15] and more recently by means of comparative twin and sibship studies.[10] A comparison of the histories of one-egg and two-egg twin pairs reveals that the striking similarities in general health records, intellectual performance, basic patterns of adjustment and social achievements, observable in one-egg pairs, tend to express themselves throughout life and generally exceed those seen in senescent two-egg twins. It is especially significant that the observed differences are clearly correlated with variable capacities for adjustment to old age.

As to the total longevity scores, the mean intra-pair difference in the length of life of one-egg twins is approximately one-half that of two-egg pairs of the same sex.[9] Only if the latter are alike in the ability to survive till the eighth decade of life, can the remaining variations in their life span be expected to be nearly the same as those of longevous one-egg pairs. Apart from certain sex differences in the degree of adaptive plasticity, corresponding trends toward a similar intellectual test performance in later life can be seen in all test scores measuring abstract intellectual function.[5] One is justified to conclude, therefore, that significant variations in the capacity for maintaining a state of physical and mental

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SIMILAR LIFE CONDITIONS—DIFFERENT GENES

Figure 9

The A. twins at ages 21, 56 and 65 years. The pattern of aging, physical and mental, was quite different in spite of very similar environment.

health throughout life are based on genically determined gradations of biological survival values and adaptive potentialities.

3. The third way, in which heredity may change the degree of adjustment to aging, is by means of interacting in the etiology of distinctly *pathological conditions peculiar to senescence*. In this group, it is to be expected that clearer genetic evidence is available for specific and relatively rare disorders, distinguished by prematurity and the presence of gross but circumscribed lesions, than it is for the more common types of senile dementia. Any processes involving a progressive loss in homeostatic capacities are relatively easy to classify and to trace in families, if the loss develops dramatically and long before its time and if it occurs in persons who originally appeared to be endowed with a satisfactory capacity for health and longevity. Under conditions of this kind, one may not even find it too difficult to think in terms of specific disturbances produced by major mutant genes, that is, by the single-factor type of inheritance.

Genetic Basis for Senile Decline

In fact, theories of simple dominance or recessiveness have been advanced not only for the three special types of presenile brain atrophy described by Pick,[1,4] Alzheimer[12] and Jakob and Creutzfeldt,[3,13] but also for the clinical disease entities of essential hypertension and cerebral arteriosclerosis[16] and for the total group of disorders called *senile dementia*.[2,14] However, since the supporting evidence regarding familial occurrence and symptomatological consistency can by no means be considered as conclusive, the need of extensive genetic studies is apparent for most of these syndromes. Genetically it is still quite likely, for instance, that the inheritance of arteriosclerosis may be modulated by a number of different genetic entities, each of which would seem to be controlled by a specific genetic mechanism. This would be analogous to the complicated modes of transmission assumed in diabetes mellitus.

The likelihood of a *multifactorial* etiology and *polydimensional* organization is especially strong with respect to those common but pathogenetically diversified psychotic syndromes in the involutional and senile periods of life, which are characterized by agitated anxiety and a combination of depressive and paranoid features. It is indicated by the distribution of involutional and senile psychoses in a sample of over 3,000 twin families that the expectancy rates for the cotwins and siblings vary according to both the psychiatric classification of the original twin cases and the degree of consanguinity to the particular type of index case. However, apart from a marked increase of

THE DIZYGOTIC B. TWINS AT AGE 83
Figure 10

Inseparable until one had to be hospitalized for senile deterioration, the B. twins differed greatly in physique and personality.

involutional and senile psychoses in the sibships of the given groups of index cases, there is an excess of involutional psychoses in the families of schizophrenics, which corresponds with an increased incidence of schizophrenia among the blood relatives of involutional cases.[8] This excess seems compatible with an expected increase in the schizophrenia rates for blood relatives of aged persons, who are distinguished by schizoid personality traits and, therefore, may be identified as heterozygous carriers of the predisposition to schizophrenia. By contrast, the incidence of schizophrenic phenomena in the families of senile index cases has been found to be negligible.

Apparently, the development of an *involutional* psychosis requires not only the capacity for survival until the later years of maturity, but also a certain combination of causative elements including progressive impairment of general adaptability, cumulative emotional strain and insecurity due to increasingly conspicuous signs of aging, and the possession of some special personality traits such as rigidity, compulsiveness and oversensitivity, frequently associated with a schizoid personality structure or a mild schizophrenic defect. The long-range effect of these traits is toward a reduction of the adjustive plasticity of aging persons and, thereby, toward an impairment in their faculty of adaptation to involutional changes.

In the group of *senile* psychoses, the genetic component implying an indirect relationship between a diluted and late-developing type of psychotic reaction in the aged and the genetic entity of schizophrenia seems to be largely replaced by an age-specific intensification of long-existing but minor deficiencies in general emotional adjustment. If this interpretation is correct, the search for a single genetic factor as the basic cause of *a senile psychosis* is likely to fail.

Aging in Twins

The photographs of a few typical twin pairs, taken from our sample of senescent twin index cases, may suffice to demonstrate the different capacities of the two groups of twins for displaying intra-pair differences in the degree of aging. For instance, in the genetically dissimilar (dizygotic) A. twins (Figure 9) very similar life conditions did not preclude the development of pronounced dissimilarities in the physical and mental signs of advancing age. Likewise, the B. twins (Figure 10) were dizygotic and never separated prior to the hospitalization of one of them. Nevertheless, they were distinguished by the fact that the younger looking twin, described as sociable, placid and kind, remained active and well-adjusted throughout her senium, while the taller and more intelligent twin sister, who had always been moody, ill-tempered and vindictive, developed a depressive-paranoid psychosis around the age of 75.

The three persons shown in Figure 11 were three-egg triplets who lived in different continents for many decades. The youngest looking member of the set claimed to have made a major contribution to the preservation of her youthfulness by coming to this country in her teens.

ON THEIR 85th BIRTHDAY

Figure 11

These three people—each living in a different country—were all 85 years old when these photographs were made. They are three-egg triplets. The youngest looking of the three attributes her success in staving off the ravages of time to coming to the United States more than 65 years ago.

By contrast, entirely different environmental circumstances had little effect on the vigor and general appearance of the C. twins (Figure 12), a one-egg pair of physicians. One of them was married for many years and had his practice near New York City, while the other preferred to remain single when he became a successful physician in a rural district.

In the monozygotic D. twins (Figure 13), prolonged separation with residence in different countries did not interfere with the development of equally severe symptoms of cerebral arteriosclerosis at practically the same time. In this pair, too, one partner remained single and the other was married for many years.

The one-egg H. twins (Figure 14) showed remarkable similarities in the type and symptomatology of a senile psychosis, in spite of striking differences in their life histories. They were 17 years old when they were separated through the marriage of one of them to a local grocer. The other twin remained single and supported herself as a housekeeper in another state until, at the age of 73, she developed an acute anxiety state with mild confusion and agitation and with vague ideas of reference and persecution. A few months after she had died of cardiac decompensation in a mental hospital, the survivor began to duplicate her twin's history in practically every respect.

Conclusion

In conclusion, it may be stated that an analysis of fully recorded twin histories cannot fail to be helpful in investigating the numerous challenging problems posed by severe maladjustment to aging and old age. *Prophylactically* it may be inferred that the most promising contribution to the prevention of an involutional or senile psychosis would seem to rest upon the establishment of adequate emotional adjustment before senescence. Further progress in the *treatment* of senile disorders is certain to come from an improved knowledge about the nature and interaction of age-susceptible personality traits prone to disarrangement by a reduced homeostatic (adaptive) plasticity and of those gene-specific biochemical phenomena controlling growth and decline.

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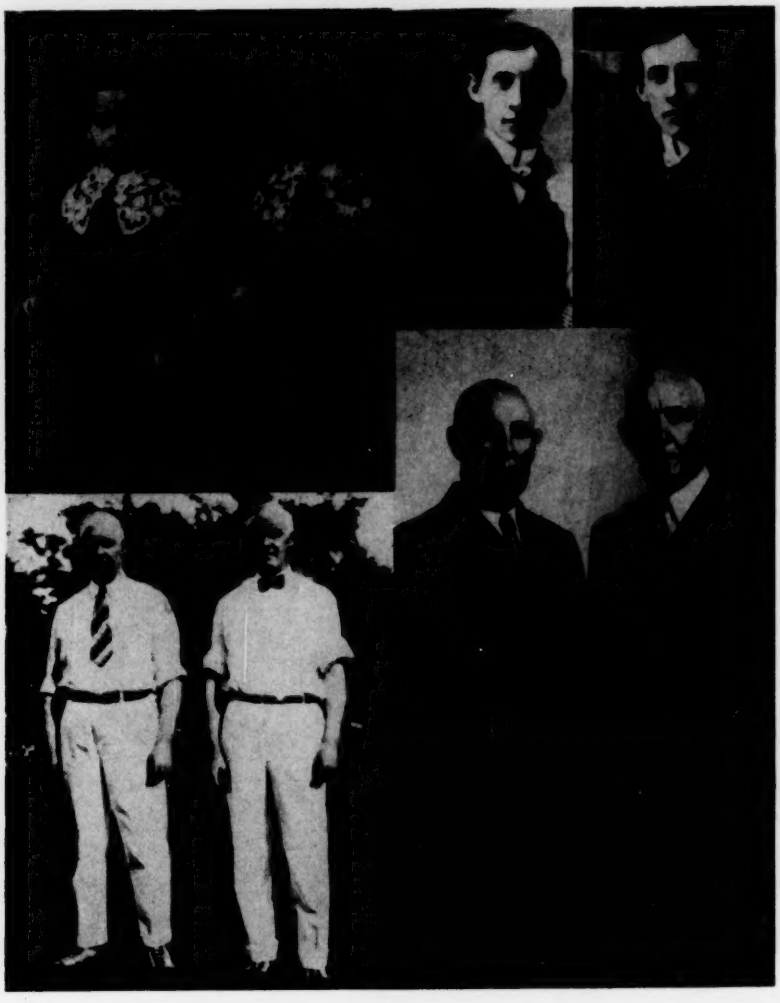

BACHELOR AND BENEDICT

Figure 12

The C. twins at the ages of 4, 16, 52 and 68 years. Both are physicians. One married and practiced in the city. The other remained single and spent his life as a country doctor. They are still not easily told apart.

THEIR LIKENESS PERSISTS IN SEPARATION

Figure 13

The D. twins at the ages of 6, 14, 60 and 68 years. Living in different countries—one married, the other single—they both displayed symptoms of severe hardening of the cerebral arteries almost identically.

GROCER'S WIFE AND SPINSTER

Figure 14

The H. twins at the ages of 1, 5, 25 and 70 years. Three years after the last photograph was made, the unmarried twin suffered a mild senile dementia. Shortly thereafter her married sister followed almost exactly the same pattern of decline and death.

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THE EUMELANIN HORSE: BLACK OR BROWN?

(*Continued from page* 76)

scheme of the author would make dominant white (W) an all white mutation (probably lethal when homozygous like the similar mutation in mice). The designation W'' W''' for genes effective in producing types of pinto (pied) with more or less extended areas of white, would seem to imply that these are regarded as alleles of dominant white. But it is doubtful if this is the author's real intention."

In several passages of the book and signally in the summary—given in the form of an extended table with the names in five countries of 24 horse colors and the corresponding 24 genotypes as postulated—the three mentioned symbols are not printed as given by Castle but as W, W', W''; Roman letters—by contrast to the Italics of point mutations—being taken as symbols of chromosome rearrangements.

In the book I endeavored to foster the hypothesis that the series of "dominant white spotting" ending with the dark-eyed whites, is in the horse—and maybe in other mammals—the outcome of position effects, perhaps through different degrees of heterochromatization in a definite region of one chromosome: the symbols W, W', W'' being allotted, with deliberate vagueness, to three such degrees or "states." That would account for the frequent lethality of the WW combination making true breeding for white color in horses an almost hopeless enterprise, as the Kings of Denmark found out at Frederiksborg.

So much for the points I do not find truly presented in the review. In such a vast field, it was not to be expected that Prof. Castle would agree with every opinion of mine; he gives grounds for his disagreements. There is not space now to take every point in turn, but (after careful examination of the reviewer's arguments) I think the reasoning, as developed in my book, still holds. Perhaps I might be permitted to mention how curious I find the reason Castle gives *against* my interpretation of the "diluting" gene as a mutant in the *C* series, namely, that such a mutant would be expected to exert stronger effect on yellow than on black pigment. A comparison of the buckskin phenotype with its corresponding "intense" (that is, the bay) shows the black pigment practically unaffected, whereas the other is reduced from red to pale yellow, a striking contrast that is peculiar to the buckskin coat. Black pigment being either absent or inconspicuous in chestnuts, the contrast in their dilutes canont be so marked. But the black spots of some chestnut horses (many Thoroughbred descendants of *Stockwell* for instance) appear as well in the respective dilutes, a fact that could be testified to by any breeder of Palominos.

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INHERITANCE OF A SKULL DEFECT IN COCKER SPANIELS*

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SINCE early in 1944 the author has maintained a small kennel of cocker spaniels at Louisiana State University for the purpose of investigating the inheritance of as many characteristics as possible. A number of breeders have furnished data on characteristics which have occurred in their kennels but have not shown up in the author's dogs. In June of 1949, a breeder wrote to the author describing what she called "open soft spots" in a number of closely inbred cocker spaniel puppies.

The cranial sutures, which are present in normal newborn cocker spaniel puppies, are so small that only the most careful examination with the finger nail can detect them. The author is not able to feel them with the finger tip. By the time the normal cocker spaniel puppy is about two weeks of age there is no evidence of this cranial crevice. On the other hand the breeder states that the "soft spot" is about one-eighth of an inch wide and one-fourth of an inch long which she can feel with her finger tips. She states that these "soft spots," which normally disappear in her other puppies soon after birth, become even more apparent as the cranial bones grow — in fact, increased to about one-fourth of an inch in width.

These defective skulls are apparently developmental calvarium defects or possibly pathological fontanelles which are "persistent" according to Worthman (*in lit.*).

Similar Skull Defects Previously Reported In Other Mammals

The "Catlin Mark," an inherited defect in the parietal bones in man, was reported by Goldsmith[1]. In most cases, it was claimed that the original single large elongated opening of the young later developed into two more or less circular ones by the ossification in the median region, thus appearing as "eyes in the back of the head."

Nordby[4] reported a defect in swine which he termed a brain hernia on the front part of the skull, and with skull openings in the defective pigs. The defect was under observation in five purebred herds for four years. Sufficient data were available to permit the defect to be recognized as hereditary, and, in most cases, it was lethal inasmuch as only very few affected pigs survived. The pigs that did live were susceptible to epileptic fits. A defect that is apparently identical has been described by Hughes and Hart[4] in Poland China swine, and they stated that apparently the defect appeared only when the offspring received the recessive factor from both parents. This defect is apparently the same as the one which Williams[6] reported as "Hernia Cerebri."

Keeler[3] described a similar defect in mice which he called "Parted Frontals." The cranial variation seemed to be associated with a blood vessel piercing the skull at an angle, passing to the exterior between these bones. It was pointed out that the skull defect was apparently inherited as a dominant character.

Shaw[5] reported two Holstein-Friesian calves which had an unusual skull defect consisting of an opening affecting both the frontal and parietal bones with brain tissue protruding out of the opening. In both cases the pia mater and dura mater membranes were missing. These two calves had the same sire and dam. Since the dam of these calves had previously given birth to a normal calf from a different sire, the facts seemed to indicate that this skull defect might be hereditary. He stated that it was similar, if not identical, to the skull defects in swine, humans and mice reported by other authors.

According to Worthman (*in lit.*), Ellenberger and Baum stated: "In contrast to man, in most of our domesticated animals true fontanelles do not occur. In dogs, however, defects of the calvarium frequently occur which remain throughout life and not just for the first three to six weeks. Müller contends that fontanelles in dogs are pathological."

Skull Defect in the Cocker Spaniel

A breeder of cocker spaniels corresponded with the author on several occasions concerning her bitch which had produced five litters of puppies by a dog and his son. This breeder

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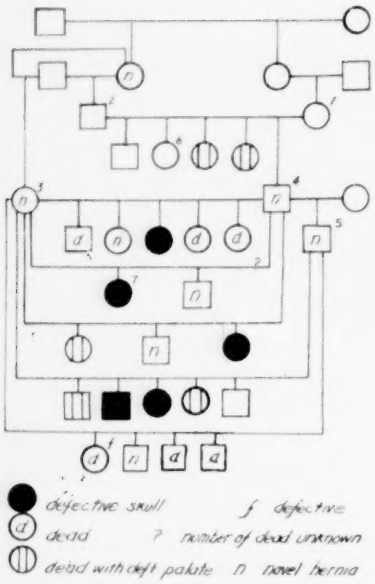

DEFECTIVE PUPS HIGHLY INBRED

Figure 15

The close inbreeding practiced in this strain of cocker spaniels appears to be increasing the concentration of a gene or genes for skeletal and other defects. The economic importance of this build-up in this hereditary defect is indicated by the fact that of 20 puppies only five lived to a "salable" age.

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wrote that her bitch, female No. 3, gave birth to from three to five whelps in each litter with only one to three alive. The next day one puppy would be strong and the others would be weak — smacking, but not nursing. The breeder used every resource in an attempt to save them so that subsequently she could find out what was wrong. The longest that she was able to keep one alive was seven weeks at which time, by palpation, the "open soft spot" in the skull of this female puppy was still evident. This one, which is female No. 7, did not grow normally as noted in Table I when compared to the strong one; in fact, she was a dwarf in comparison with her litter brother. According to the breeder, female No. 7 had passed through all the normal stages of physical and mental development of a seven weeks old puppy except that she just failed to grow at the proper rate. Then female No. 7 died suddenly. The breeder thought that her death was due to "tussling" with her brother and that he might have "bumped" her, as no marks were apparent on her. She found the puppy dead just two hours after she had seen her alive and "happy." (The author thinks that 94 ounces is about what a well developed seven weeks old cocker puppy should weigh.) Each one of these "fading" puppies had an "open soft spot" on its head. The veterinarian told the breeder that it happened periodically and that he had never heard of a puppy with this skull defect surviving.

Among the dead puppies there were two malformations in two of the litters of which one was the puppy noted under dead as defective (Table II) and the other was not recorded as defective dead in the whelping book, but only as dead.

The two sires and the dam all had navel hernias. The five puppies that lived all developed the same type of navel hernias as found on their parents. When male No. 4, who had a hernia, was mated to bitches without hernias, the breeder could make only general statements. In some litters there were none, others only one, and in one litter there were two of them.

The three animals, female No. 3, male No. 4, and male No. 5, are closely related as noted in Figure 15. The mating of female No. 1 to male No. 2, whose dams were litter sisters, produced a litter of five puppies having three females and two males, one (No. 4) of which had consistently produced this defective skull in every litter when mated to female No. 3

TABLE I. Development of "dwarf" female No. 7 with defective skull, and her normal litter brother.

Age	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	28	35	42	49	days
♀	9	9	9	9½	9	9½	9½	9½	9	9½	9½	10	10½	10½	11	11	11	11½	12	17½	22½	30	40	ounces
♂	8	10	12	14½	16½	18	19½	22	23	23	24	26	28	31	31	33	34	35	37½	49	60	75	94	ounces

TABLE II. Data of five litters of inbred cocker spaniel puppies taken from breeder's whelping record book.

	Matings	Date Whelped	No. in Litter	Whelped Dead and with Cleft Palates	Whelped Dead	Skull Defect and Died	Lived and had a Navel Hernia
1.	♂ No. 4 × ♀ No. 3	3/29/45	5(1 ♂, 4 ♀)	0	3(1♂, 2 ♀)	1 ♀ (lived 2 days)	1 ♀
2.	♂ No. 4 × ♀ No. 3	1/1/46	?	?	?	1 ♀ (No. 7 lived 7 wks.)	1 ♂
3.	♂ No. 4 × ♀ No. 3	8/28/46	3(1 ♂, 2 ♀)	1 ♀	0	1 ♀	1 ♂
4.	♂ No. 6 × ♀ No. 3	2/19/47	5(3 ♂, 2 ♀)	2(1 ♂, 1 ♀)	0	2(1 ♂, 1 ♀)	1 ♂
5.	♂ No. 5 × ♀ No. 3	8/10/47	4(3 ♂, 1 ♀)	0	3(2 ♂, 1 ♀ defective)	0	1 ♂

who is a full sister of his sire (No. 2). Concerning the three litter sisters of male No. 4, two of them were whelped dead and in the third one, female No. 6, the breeder fought convulsions for six months. The veterinarian decided that this was a case of epilepsy and put the bitch to sleep. A study of Table II tells the same story of a high percentage of dead puppies whelped in four litters when male No. 4 or his son were bred to female No. 3 who was closely related to them. In the single litter where the breeder failed to record the dead ones, she stated that there were dead ones in it just as in all the other litters. Among all these dead puppies there were some which were apparently normal phenotypically while others had cleft palates. Besides the ones whelped dead in every litter there were the ones having the defective skulls which lived only from a few hours to a few days except the one female (No. 7) which lived seven weeks. Out of over twenty puppies only five lived to be of "salable" age.

Even the son (No. 5) of male No. 4 when mated to the same female (No. 3) produced the same phenomenon—dead puppies and the defective skull.

Summary

1. This defective skull in cocker spaniels is apparently inherited as a recessive which is lethal.

2. The author suggests that since there are many puppies whelped dead in these inbred cocker spaniels that there is a possibility of lethals other than the defective skull.

3. Apparently the cleft palate is inherited as father and son both produce it when mated to the same female, but these data plus other data which the author has, do not suggest a mode of inheritance.

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LENGTH OF THE GESTATION PERIOD IN BEEF CATTLE

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STUDIES concerning lengths of gestation periods for the breeds of beef cattle have shown that there is a statistically significant difference between breeds. The differences have varied from study to study, but the gestation lengths for Herefords and Shorthorns have been found to be longer than those for Aberdeen-Angus.

Lush[4] found the average gestation period of cattle to be 282.1 days with a standard deviation of about five days for individual gestations when 27,810 gestations were studied. These data included various breeds from many locations.

Johnson, as cited by Shrode and Lush,[5] reported that Aberdeen-Angus gestation periods were slightly (about 2.5 days), but significantly, shorter than those for Shorthorns and Herefords in the South Dakota State College herd. Analysis of variance between sires and dams indicated a small repeatability for cows (four to seven percent of the variance) but a much smaller effect (statistically quite insignificant) of the sire of the calf.

Livesay and Bee[3] found Aberdeen-Angus calves of both sexes to have an average gestation period of 282.5 days, the males averaging 282.7 days and the females, 282.2 days. They also found a longer gestation period of 5.8 days (statistically significant) for the beef breeds when compared with the dairy breeds.

Gerlaugh, *et al*[1] reported an average gestation length of 276.47 days for purebred Aberdeen-Angus calves and 286.28 days for purebred Herefords with the crossbred calves being carried in utero an intermediate length of time. The Hereford-Angus calves were carried 281.98 days, whereas the Angus-Hereford crossbreds were carried 283.30 days.

For the Indian breeds, Littlewood[2] gave gestation lengths of 289.3 days for Gir, 292.2 days for Nellore, and 291.9 days for Guzerat.

Data Used

The 551 gestation periods included in this study are those for cows in the purebred Hereford, Aberdeen-Angus, and Shorthorn herds

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owned by the Department of Animal Husbandry, A. & M. College of Texas, College Station, and the Hereford and Brahman × Hereford crossbred herds at the East Texas Pasture Station, Lufkin, Texas. Included in the data are 229 gestations for Hereford calves, 195 for Aberdeen-Angus calves, 63 for Shorthorns, 44 for Brahman × Hereford crosses, and 20 for calves out of Hereford cows sired by Brahman × Hereford bulls.

Results and Discussion

The average lengths of gestation by breed and sex are shown in Table I.

Analysis of variance revealed highly significant differences in length of gestation among different breeds. The average for the Aberdeen-Angus was almost six days shorter than that for Herefords; and the average for Shorthorns was between the two, being almost four days shorter than that for Herefords. The Brahman influence apparently resulted in slightly longer pregnancies for the Brahman × Hereford crossbreds than for the purebred Herefords.

TABLE I. Average length of gestation for purebred and crossbred calves.

	Both Sexes		Males		Females	
	No.	Avg.	No.	Avg.	No.	Avg.
Hereford	229	285.22	111	285.44	118	285.00
Aberdeen-Angus	195	279.48	96	279.10	99	279.66
Shorthorn	63	281.32	33	283.60	30	278.80
½ Hereford-½ Brahman	44	286.32	20	286.90	24	285.82
¾ Hereford-¼ Brahman	20	285.70	11	287.36	9	283.66

TABLE II. Analysis of variance of gestation periods for 551 calves.

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square
Total	550	24935	
Between breeds	4	4321	1080.5*
Between sires within a breed	17	2763	162.6*
Between dam age groups within sires within a breed	112	4950	37.5
Between sexes within dam age groups within sires within a breed	85	2559	30.1
Between individuals within sex and dam age groups within sires within a breed	312	10340	33.1

*Highly significant

The sires included in the study were 10 Herefords, five Aberdeen-Angus, three Shorthorns, two Brahmans, and two Brahman × Hereford crossbreds. The differences between these sires within a breed were highly significant. The greatest ranges in average gestation lengths of their progeny were among the Aberdeen-Angus, Hereford, and Shorthorn bulls with ranges of 11.05, 7.31 and 6.97 days respectively. The small number of calves sired by some of the bulls undoubtedly accounted for much of the variation.

Differences between age groups of dams within sire were not statistically significant. The means of the gestation lengths for the different dam age groups failed to indicate any noticeable tendency for length of gestation to increase with age of the dam. The sex difference was not significant. The male calves were carried slightly longer than the females except in the Aberdeen-Angus where the heifers had a slightly longer period.

Summary

The data used in this study were the records of 551 gestation periods representing five breeds and crosses of beef cattle.

A highly significant difference in length of gestation was found between breeds, and between sires within a breed.

There was no significant difference in length of gestation due to sex of calf even though the male calves were usually carried in utero slightly longer than were the females.

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SUGARCANES IN THE FAR EAST

Some Cytogenetical Considerations

A. MORIYA

College of Agriculture, Utsunomiya University
Utsunomiya, Japan

SOME thousands of varieties of sugarcane have been successfully obtained by cross breeding. The dramatic rescue of the Javanese sugar industry from the serious "sereh" disease by the development of improved resistant varieties, was a classical example of scientific plant breeding. Most of the commercial varieties cultivated in the world today may be said to have originated from those same Javanese canes which reestablished the Javanese sugar industry. What was the basic material from which these excellent varieties were derived?

Javanese seedlings are often referred to as the "Kassoer strain" or the "Glagah strain," Kassoer or Glagah (*Saccharum spontaneum*) having been used in their development. Kassoer was found growing wild on Mt. Tjeremai, Java. Morphological and cytological analysis of this cane led to the conclusion that it was identical with the artificial hybrid Black Cheribon × Glagah, which supposedly originated from this cross in the natural condition.[3,4,5] It must be noted here that chromosome doubling on the female side occurred in every case in the cross Black Cheribon (*S. officinarum*, $2n = 80$ ♀) × Glagah (*S. spontaneum*, $2n = 112$ ♂). This resulted in all the F_1 hybrids having 136 chromosomes—40 more than the sum of the haploid chromosome number of both parents. As a consequence of their polyploid nature, F_1 plants have juice with a high sucrose content. They have thick stems, are disease resistant, and they display the vigorous growth habits of the wild cane.

Subsequently many *S. spontaneum* varieties were crossed in other countries with *S. officinarum*. Without exception all the F_1 hybrids had 40 more chromosomes than the expected number[9]. This phenomenom is very important when *S. spontaneum* is used for sugarcane breeding. For example, in India descendants of F_1 ($2n = 112$) primitive cane (*S. officinarum*, $2n = 80$) × wild cane (*S. spontaneum*, $2n = 64$) are used in cross breeding.[1] In the Philippines Hind's Special ($2n = 120$) and Toledo cane ($2n = 120$) are believed to have resulted from the natural cross of native primitive canes (*S. officinarum*, $2n = 80$) and native wild cane (*S. spontaneum*, $2n = 80$). They are used to produce other crosses.[7] The variety O 1744 originated from the cross Ardjoeno (*S. officinarum*, $2n = 80$) × Glagah Tabongo (*S. spontaneum*, $2n = 80$) from North Celebes. It showed $2n = 120$. In this case also it is reasonable to suppose the female gamete is diploid.[4]

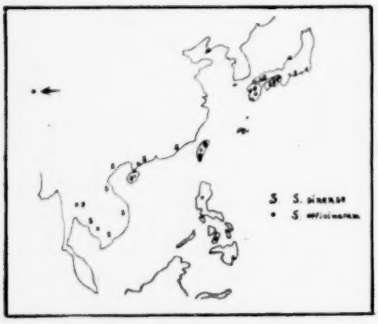
Another utilization of the wild sugarcane is to use it as material for breeding cold-resistant sugarcane. This is a comparatively recent development, studied mainly in the United States, where many *S. spontaneums* from high latitudes have been assembled and crosses made between them.[2,6]

In this paper the strain preferred for breeding in the Far East will be discussed, based mainly upon my earlier studies, especially in Formosa where, before World War II, sugarcane breeding was most actively practiced. The Far East is defined as extending from Thailand to China, Japan, Hainan Island, and the Philippines, excluding Borneo where a modern sugar industry has not been developed, and Java, which is better discussed elsewhere.

Kinds of Native Canes in the Far East

1. *Cultivated Canes*

Primitive canes in the Philippines are considered by Okade[13] to belong to *S. officinarum* ($2n = 80$) and my study[10] confirms this. All



DISTRIBUTION OF SUGARCANE SPECIES

Figure 16

This outline map of southeast Asia delimits the known distribution of the two species which have most contributed to the development of superior varieties.

the primitive canes in Thailand and Indochina have been thought to be *S. sinense* ($2n = 116$-118).[13] But some varieties that are morphologically and karyologically *S. officinarum* ($2n = 80$) are also found there. In southern China, Formosa, and Japan a few *S. officinarum* varieties coexist with *S. sinense*.[10] Most of the cultivated canes in Japan proper are Tekcha-like; narrow, leaved, slender varieties, and perhaps *S. sinense*. In Hainan Island there are many primitive canes. Among 11 varieties which I studied, at least one was assumed to be *S. officinarum* judging from the external morphology and the measurement of stomata size in the lower surface of the leaves (Moriya unpublished). *S. officinarum* is in the same way found in Japan proper, although on a small scale of cultivation. Figure 16 shows the above distribution.

2. *Wild Canes*

S. spontaneum is the only wild sugarcane found in this area. *S. narenga* is not genuine *Saccharum*,[11] so it will not be discussed here. *S. spontaneum* is known by taxonomists to be widely and abundantly distributed in Indochina, Thailand, and the Malay Peninsula. Chromosome numbers have been studied in forms from the Philippines, Formosa, Amami Oshima, and Japan proper.

In my opinion, *S. spontaneum* plants in the Philippines, Hainan, and Formosa are of different kinds judging from the morphology, chromosome number, and differences in flowering season. *S. spontaneum* in Formosa, Amami Oshima, and Japan are alike morphologically and probably represent a common distribution.[11] Forms from Amami Oshima and Japan proper developed no inflorescence when transplanted to Formosa suggesting a slight difference from the Formosan *spontaneum*. *Spontaneum* plants from Okinawa main island were brought here last spring and are growing in our greenhouse. These resemble morphologically and in height the var. *Roxburghii* ($2n = 96$) in Formosa, although leaves and stems are somewhat more slender. *Spontaneum* in Japan proper has a diploid complement of 112 chromosomes, the var. *genuinum* in Formosa shows a quite similar appearance.

Origins of the Improved Sugarcane Varieties in the Far East

Systematic cross breeding was practiced only in the Philippines and Formosa in this area.

Philippines

Around 1935, POJ 2878 occupied nearly half the acreage of the sugarcane plantations in the islands, and Badila about 19 percent.[13] At that time the Philippine-bred varieties had not come into cultivation. Recently, PSA 7 and PSA 14 increased in acreage. It is worthwhile to note that they are the descendants of Talahib ($2n = 80$) which is wild cane (*S. spontaneum*, $2n = 80$) in the islands. This method of breeding is the same as in Java, where POJ 2878 and other superior varieties were bred. To cross the best acclimated primitive canes with the wild cane of the same land is assumed to be the ideal method.

Formosa

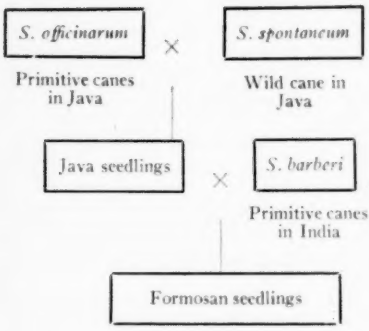
Acreage of each variety cultivated in Formosa, before and at the end of War II, is shown in Table I.

From Table I it is clear that during the nine year period POJ varieties from Java dropped from nearly 100 percent to about 51 percent; while the Formosan F-varieties increased from 0 percent to 48 percent. Parentage of new varieties from F 105 to F 133 which were bred by Japanese breeders are shown in Table II.

These 29 varieties shown in Table II are classified in Table III according to the kinds of species crossed.

Among the 29 varieties listed in Table III only six, (F 105, F 107, F 108, F 109, F 110, F 113) are listed in Table I. These are the newest and most accurate statistical data available. The four most promising varieties (F 108, F 109, F 110, F 113), which constitute 47.68 percent of the total in cultivation, are shown in line 1, Table III. They are hybrid descendants of three species, *S. officinarum* *S. spontaneum* (Java), and *S. barberi*. F 118 is very promising, but it was not cutlivated for the mill up to 1945.

From the above discussion, the pedigree of the promising seedling varieties of Formosa can be diagrammed:



This pedigree shows that for best results the tropical Java varieties are crossed with subtropical Indian canes; in other words, the disease resistance and stem rigidity of *S. barberi* are taken into the Formosan canes, which is of significance in such a wind-swept land.

Among the Java seedling varieties derived from *S. officinarum* × *S. spontaneum*, POJ 2725 and POJ 2878 were most frequently used in crossing in Formosa. Twenty-four of the 29 varieties listed in Table III are descended from these two POJ varieties.

1. 9 varieties, *Descendants of POJ 2725:* F 105, F 108, F 113, F 115, F 125, F 126, F 127, F 132, F 133.
2. 15 varieties, *Descendants of POJ 2878:* F 106, F 107, F 110, F 111, F 114, F 116, F 118, F 119, F 120, F 123, F 124, F 125, F 126, F 127, F 128.

It may be easily recognized that the good Formosan seedling canes raised before 1945 are almost all the descendants of either POJ 2725 or POJ 2878. POJ 2878 which is called "the wonder cane," was once the leading variety in Java. It is not satisfactory for direct sugar production in Formosa, although it is used a good deal as crossing material.

Cane-sorghum hybrids are estimated as of no value for the primary purpose intended, that is to obtain early maturing cane.[8,9] As yet, *S. robustum* hybrids have been little cultivated. However, the descendants contain hard and soft stemmed seedlings both of which are tall and seem promising material for the future. F 133 is such a strain having a stem so hard it is resistant to wind and rat damage.[12]

Breeding New Adapted Varieties

Our experience in Formosa taught us that the best basic material for breeding was *S. officinarum;* the descendants of *S. sinense*, did not give any good seedlings. *S. officinarum* itself was rarely used in crossing but hybrids having *officinarum* ancestry namely the POJ varieties of Java, gave good results. As previously described, *S. officinarum* varieties were also cultivated for a long time in Formosa, southern China, and Japan, and they are acclimatized as primitive canes. It seems possible to obtain good varieties adapted to the subtropics by crossing these *officinarum* varieties. Because it rarely develops inflorescences in the subtropics, Badila was planted in Koshun, located in the southern part of Formosa, where

TABLE II. Parentage of Formosan cane varieties accessioned after F 105.

Varieties	Parentage
F 105	POJ 2725 × POJ 1499
F 106	POJ 2801 × POJ 2878
F 107	POJ 2878 × POJ 2940
F 108	POJ 2725 × F 46
F 109	POJ 2878 × F 47
F 110	POJ 2878 × Fm 3
F 111	POJ 2878 × POJ 1499
F 112	POJ 2875 × EK 28
F 113	POJ 2725 × Fm 3
F 114	POJ 2878 × POJ 2753
F 115	POJ 2725 × F 50
F 116	POJ 2878 × EK 28
F 117	POJ 2804 × SW 111
F 118	POJ 2878 × F 84
F 119	F 58 × POJ 2878
F 120	POJ 2946 × POJ 2878
F 121	POJ 2722 × POJ 2803
F 122	Badila × EK 28
F 123	POJ 2940 × POJ 2878
F 124	POJ 2946 × POJ 2878
F 125	POJ 2825 × *Sorghum* × POJ 2878
F 126	POJ 2725 × POJ 2878
F 127	POJ 2725 × POJ 2878
F 128	POJ 2940 × POJ 2878
F 129	POJ 2875 × F 82
F 130	297/12B × Fm 3
F 131	SW 499 × F 108
F 132	POJ 2725 × F 50
F 133	POJ 2725 × *S. robustum*

TABLE I. Percentage of Harvested acreage of Formosan sugarcane varieties.*

Varieties	1935/36		1939/40		1944/45	
POJ 2714	0.03%		0		0	
POJ 2725	74.43	99.64%	30.25%	68.42%	16.01%	50.83%
POJ 2878	17.93		11.47		2.65	
POJ 2883	7.25		26.70		32.17	
Badila	0.04		0		0	
F 105	0		0.07		0.51	
F 107	0		0.32		0.18	
F 108	0	0	30.47	30.95	45.41	48.37
F 109	0		0.09		0.12	
F 110	0		0		0.12	
F 113	0		0		2.03	
Others	0.32		0.63		0.79	

*Statistics of the Formosan Sugarcane Varieties 1945.

crossing was carried out. From such experience, primitive varieties belonging to *S. officinarum* such as Aucha must be transplanted to more southern, tropical areas to enable them to develop good flowers. Day-length control should also be taken into account to promote flowering.

On the other hand, Formosan wild canes should be used as breeding material, as were the local wild canes in Java, India, and the Philippines. Natural hybrids between *spontaneum* and cultivated canes were found in Java and the Philippines where both species of cane came into flowering simultaneously. In Formosa, however, the wild canes flower from July through September; and the cultivated canes from November through January, thus leaving at least a two-month interval which never allows natural crossing. I was able to delay the flower bud formation of *S. spontaneum* (Taiwan forms) until early October by maintaining artifically a day-length corresponding to that of June 20 (13 hours and 33 minutes). At that time the bud was unable to open due to the cold climate of October (Moriya unpublished). Hence, temperature control is also necessary in Formosa when day-length control is applied to delay the flowering season of *S. spontaneum* until autumn. It may prove to be possible to cross Formosan wild canes with the existing cultivated canes by these methods. It must be noted that *S. spontaneum* matures early in Formosa which is very important in breeding an early harvesting cane.

New materials for cane breeding can be obtained in the place of Java seedling varieties useful in Formosa by crossing primitive varieties belonging to *S. officinarum* and cultivated for a long time in Formosa and its vicinity, with *S. spontaneum* originating in the same place, under controlled day-length if necessary. It appears to me that breeding new "blood" varieties like F 108, by crossing such varieties with Indian *S. barberi*, is the ideal way to obtain superior sugarcane varieties for the Far Eastern subtropics.

TABLE III. Lineage classification of the Formosan seedling varieties.

1. Hybrid Descendants of Three Species:
   S. officinarum, *S. spontaneum* (Java), *S. barberi*.
   14 varieties: F 106, F 108, F 109, F 110, F 111, F 113, F 114, F 115, F 117, F 118, F 129, F 130, F 131, F 132.

2. Hybrid Descendants of Two Species:
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3. Hybrid Descendant of Two Species:
   S. officinarum, *S. barberi*.
   1 variety: F 105.

4. Hybrid Descendant of *S. officinarum*:
   1 variety: F 122.

5. Hybrid Descendant of Three Species:
   S. officinarum, *S. spontaneum* (Java), *Sorghum*.
   1 variety: F 125.

6. Hybrid Descendant of Three Species:
   S. officinarum, *S. spontaneum* (Java), *S. robustum*.
   1 variety: F 133.

Conclusion and Summary

The Philippine manner of cane breeding is to utilize the natural hybrids between the primitive varieties belonging to *S. officinarum* and *S. spontaneum* originating in that land as breeding material. Judging from the successful results this method may be adapted also to Java and India.

Most of the superior varieties have been obtained by crossing Java-bred varieties (descendants of *S. officinarum* × *S. spontaneum*) and the Indian canes (*S. barberi*). That is to say, the best adapted canes for the Far Eastern subtropics are varieties obtained by crossing tropical varieties of Java and subtropical canes of India.

Better adapted canes are logically expected to be developed from the new breeding materials, which will be obtained by the cross between the primitive varieties belonging to *S. officinarum* cultivated in this area, and *S. spontaneum* indigenous in the same land. Finally *S. barberi* should be again crossed with these two species.

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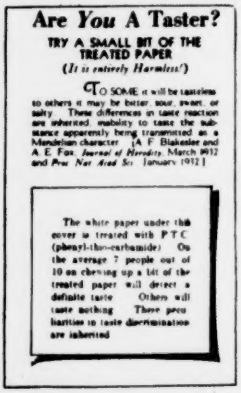
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